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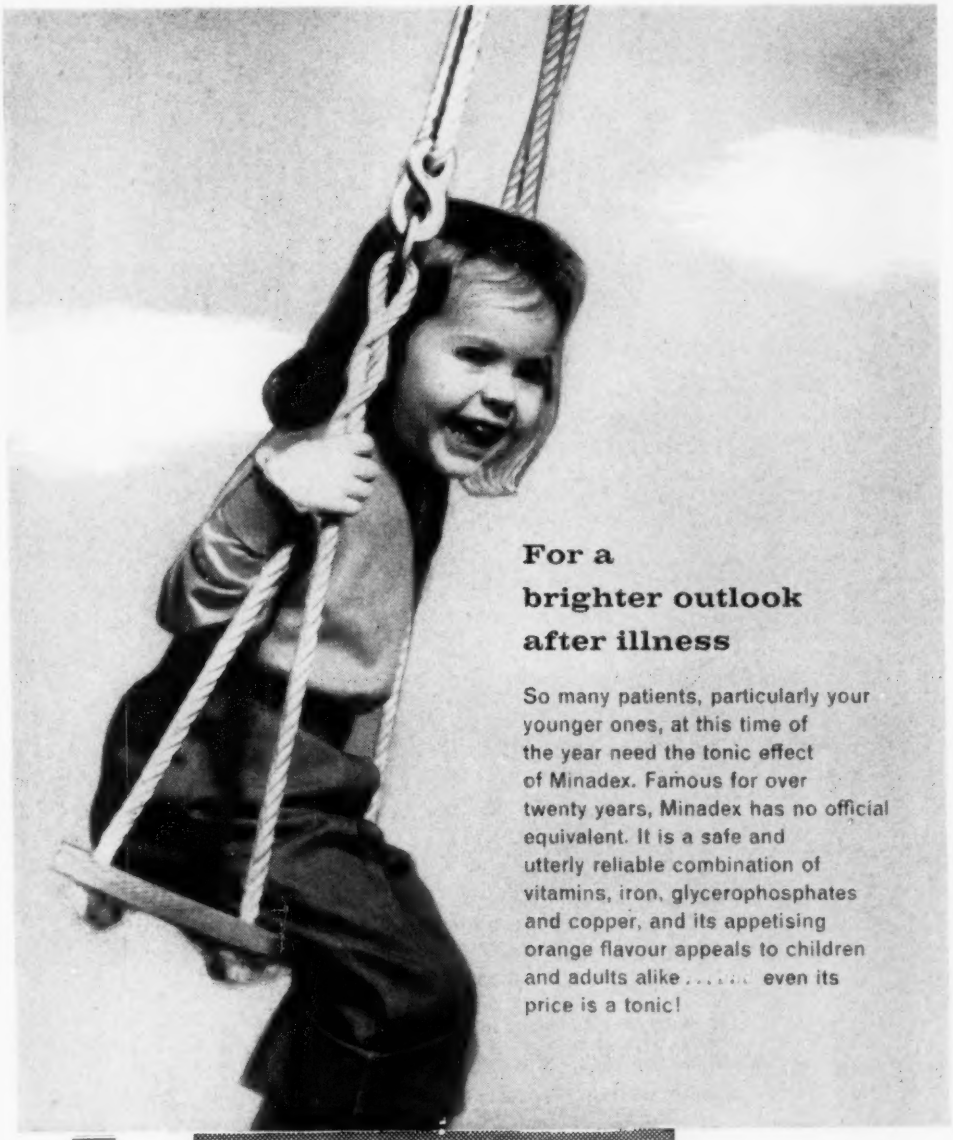

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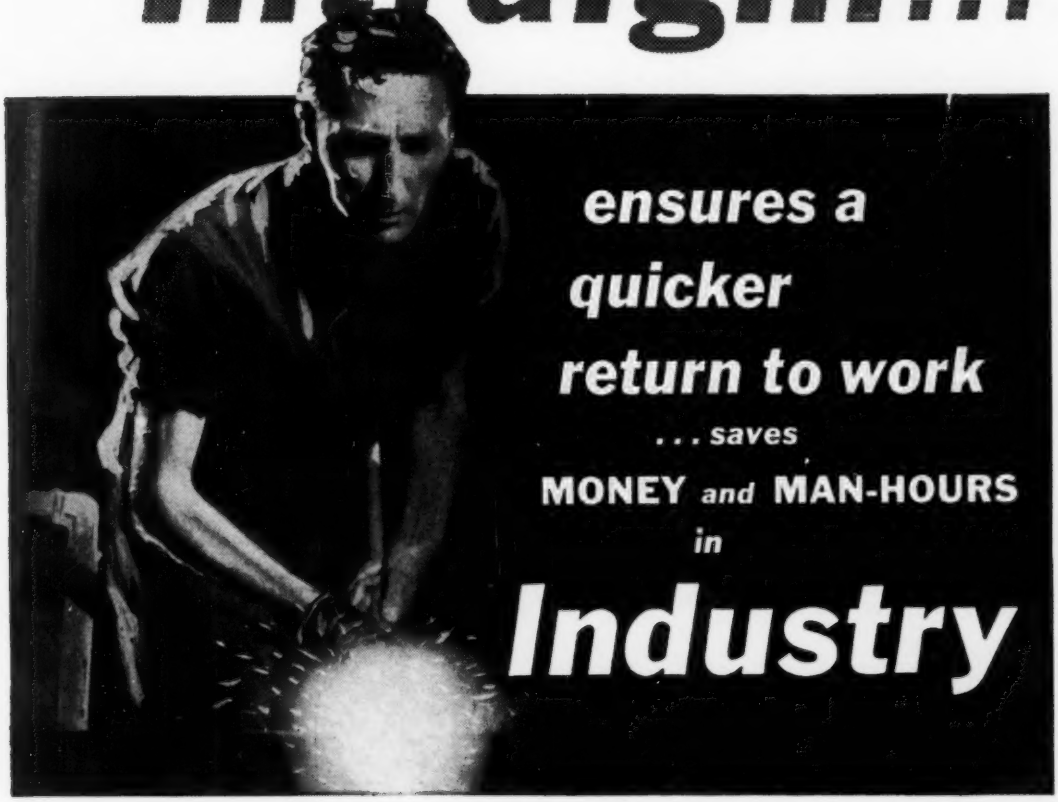

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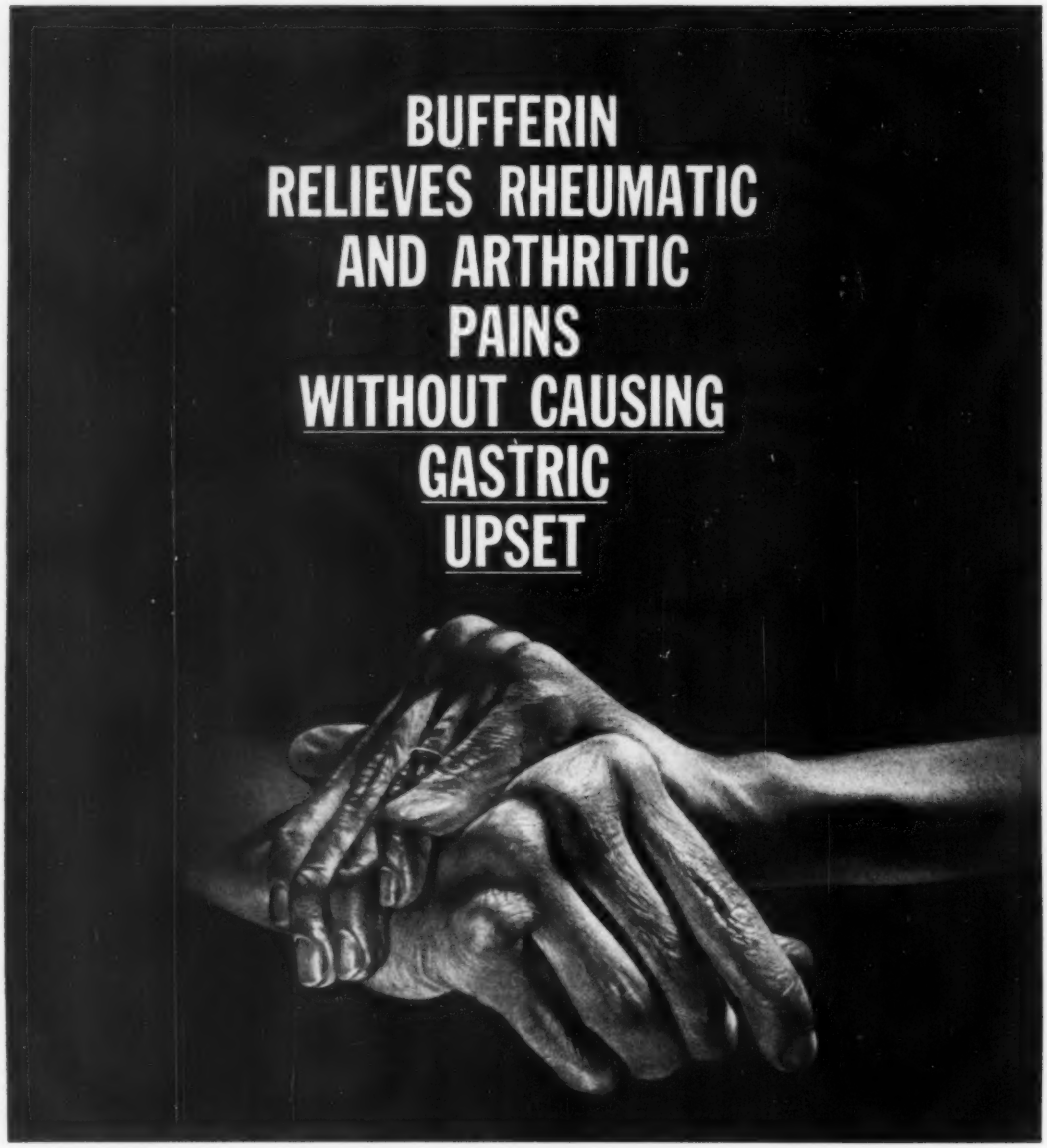

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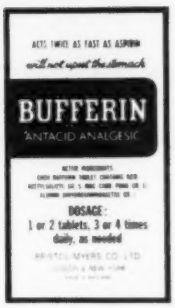

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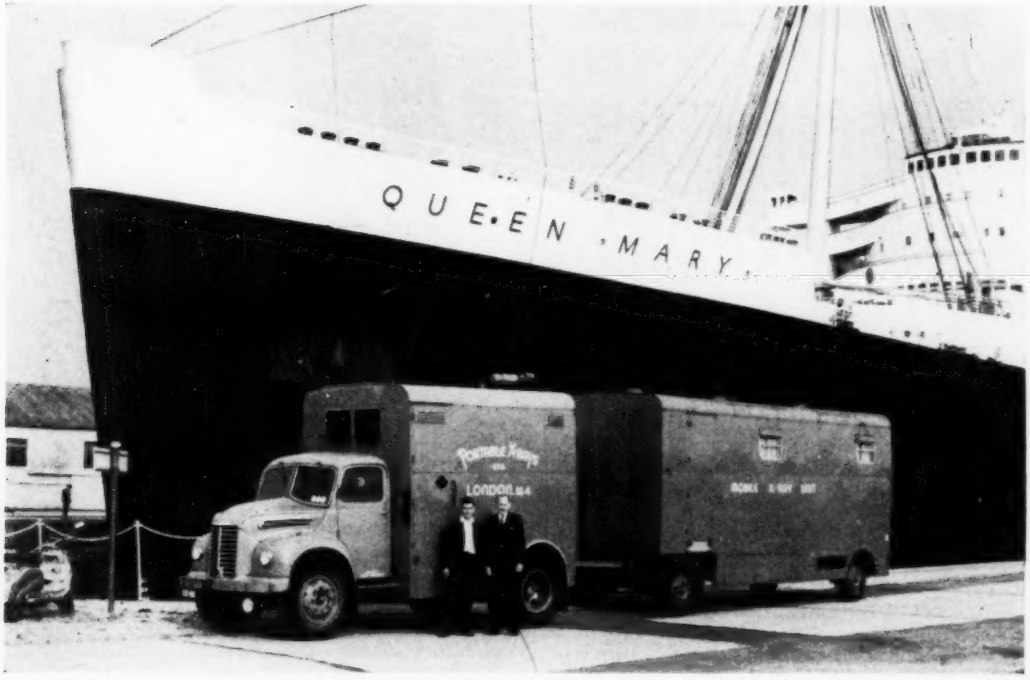

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THE EFFECT OF FILM QUALITY ON READING RADIOGRAPHS OF SIMPLE PNEUMOCONIOSIS IN A TRIAL OF *X*-RAY SETS

BY

F. D. K. LIDDELL

From the Medical Statistics Branch, National Coal Board, London

(RECEIVED FOR PUBLICATION FEBRUARY 20, 1961)

Four chest radiographs (14 in. × 14 in. postero-anterior) for each of 86 coal-miners were taken (in a trial to compare *x*-ray sets) and assessed by a number of experienced readers for both quality and pneumoconiosis. All films were developed by one technician under standard conditions so that variations in the quality of the films produced for one subject arose because of differences in the sets and in the way they were used by the radiographers taking the films. The data thus obtained allowed a study of film quality to be made (a) in relation to the subject and (b) as it affected the reading of simple pneumoconiosis.

The subjects were selected to include a high proportion whose earlier radiographs showed pneumoconiosis; they were thus substantially older than a normal colliery population.

The assessments of quality were found to be reasonably consistent both between observers and on different occasions for the same observer.

A clear tendency was found for the quality of a film to depend on the subject. Men with no radiological evidence of pneumoconiosis tended to produce films which were assessed as of better quality than those of men with pneumoconiosis, however slight. Among the latter, chest thickness had an important effect on film quality; men with thicker chests produced poorer films. The subject's age did not appear to have any effect on the quality of his film.

Film quality was found to introduce only slight biases into the reading of pneumoconiosis. Individual readers varied considerably so that, although on average the readers tended to overcorrect for technical faults, *i.e.* to read more abnormality in black films than in good ones, and less in grey, some readers undercorrected slightly.

What little evidence was available did not suggest that poor quality of films introduced any excess variability into film reading.

In 1956 the National Coal Board approved a scheme for the periodic radiographic examination of all mineworkers, to provide a biological check on the effectiveness of dust suppression in the industry, to help to eradicate tuberculosis and, of course, to offer a diagnostic *x*-ray service. Nine mobile *x*-ray units were required and a trial was held to discover which of four *x*-ray sets* produced the best films.

During the trial 86 miners at one colliery in Kent had radiographs taken on the four sets each within a few minutes. A number of experienced readers assessed the quality of each film and read them all for pneumoconiosis. The object of the trial was achieved by scoring each assessment of quality on a simple scale and by totalling the scores for the films produced on each set.

The data available allowed an examination to be made of the way in which the quality of a film is related to the subject and of the effects of film quality on the reading of pneumoconiosis. This paper discusses these examinations, after describing the trial briefly.

*Preliminary discussion with manufacturers and examination of tenders had revealed that only three manufacturers could supply and maintain appropriate equipment. Each provided for the trial one apparatus (W, XI, and Y) using one particular form of automatic exposure control; one manufacturer also offered another form of control and his apparatus using the second form of control (X2) was the fourth set in the trial.

Description of the Trial

The main intention of the trial was to compare chest *x*-ray films (14 in. × 14 in. postero-anterior)

TABLE 1
AGE AND PREVIOUS RADIOLOGICAL CATEGORY OF SUBJECTS

Two Readings of Radiological Category of 1955 Film	Age (Years)						Total
	Up to 19	20 to 29	30 to 39	40 to 49	50 to 59	60 and Over	
0, 0	3	4	6	4	4	4	25
0, 1	—	1	3	7	4	4	19
1, 1	—	—	—	3	4	1	8
1, 2	—	—	1	3	2	2	8
2, 2	—	—	1	5	4	1	11
2, 3	—	—	—	1	3	—	4
3, 3	—	—	—	—	4	2	6
PMF, PMF	—	—	—	1	4	—	5
Total	3	5	11	24	29	14	86

of approximately 100 working miners taken on each apparatus under review. Conditions were to be as close as possible to those under which the nine mobile *x*-ray units would be operating; each manufacturer was to nominate a radiographer to operate that firm's own equipment; the films were to be handled and developed in one darkroom by one National Coal Board technician using an automatic processing unit so that conditions were kept as nearly identical as possible, particularly while all the films for one subject were being processed; the assessment would be by four experts nominated by a Board Sub-committee.†

Nearly 1,400 men had been *x*-rayed at Chislet colliery in Kent in June 1955 and the films taken then, together with the readings by one observer on two separate occasions, were available. The men's ages were also known.

The subjects were chosen from the men known to be still employed at Chislet, to include a high proportion with evidence of pneumoconiosis and particularly of those whose films had been read differently on the two occasions.

The trial was carried out at Chislet in June 1957. The co-operation of the men was excellent and out of 100 selected all but 13 attended. One film was blank and all films of that subject have been excluded from this study. The ages and previous radiological categories of the remaining 86 subjects are given in Table 1. It must be remembered that the sample was not representative of a colliery population in that the proportion of men whose 1955 films were read as normal, or with little radiological evidence of pneumoconiosis, was deliberately reduced very greatly. Since the men with more advanced categories of pneumoconiosis tend to be the older men in the colliery, the sample contains a much higher proportion of older men than is representative.

Each man was allocated an appointment in random order and in only four cases was the order changed; the essential randomness was unaffected. Each man was examined on all four types of equipment in quick succession and so there were 344 films in all in the trial.

Every effort was made to ensure uniform conditions; details are given in the Appendix. The use of only one darkroom and the same technician throughout made it most probable that all films were subjected to similar conditions of handling.

Before each man had a radiograph taken, his chest thickness was measured (in cm.) at maximum inspiration at the nipple level, by one observer. It is more usual to measure chest thickness during quiet respiration and the values of this paper are, therefore, larger than usually reported in papers on radiographic technique.

It was, unfortunately, impracticable in this investigation to assess the effect of chest thickness on the 1955 films in the same way as those taken in this trial in 1957.

After the trial was completed each film was allocated a reading number in a new order which ensured that no two successive films were of the same man and that the films taken on any particular set of equipment were distributed quasi-randomly throughout the series.

Each assessor read each film independently, with all identifying marks obscured, under identical viewing conditions. He recorded for each film an assessment of its quality from the point of view of reading pneumoconiosis, scoring on the following basis:

+1: Highly acceptable, above average, very good or technically desirable.
0: Average, acceptable, technically reasonable.
−1: Below average, poor, unacceptable.

Each reader also recorded his reading of pneumoconiosis using the I.L.O. 1950 classification.

Two readers, A and B (a senior member of the Medical Research Council's Pneumoconiosis

†Later, assessments were made by a number of other readers.

Research Unit and the Medical Officer in charge of one of the Board's Research *x*-ray units), read the films on two separate occasions each. Four other readers, C, D, E, and F (another senior member of the P.R.U., a full-time experienced member of a Pneumoconiosis Medical Panel of the Ministry of Pensions and National Insurance, a consultant in industrial medicine with particular interest in and experience of chest diseases, and a clinician with previous experience of pneumoconiosis but who had not been reading radiographs for some time before the trial), read them on one occasion each. (The order in which the readers' experience is described does not necessarily correspond to the order of their code letters.)

Consistency in Assessment of Quality

The assessments of quality are summarized in Table 2. It is clear that, although there were differences between the readers in the general level of assessment, each reader placed sets X2 and X1 above sets W and Y, although there was a certain amount of interchange between X1 and X2 and between W and Y; there was no observer who placed either Y or W above either X2 or X1. It is of interest that exactly similar rankings were obtained when the films were assessed for quality by a further eight readers, including representatives of two of the manufacturers. The consistency with which sets X1 and X2 were placed highest justified their preference and, as they differed only in their methods of exposure control, the scoring, despite its apparent crudity, achieved its primary purpose. It is, of course, possible that the results were influenced either by the ancillary equipment which each manufacturer provided for his own set in this field trial or by the degree of success with which each radiographer handled his own firm's set. The reasons for the comparative failure of sets W and Y are not important since the trial was intended to assess overall performance in the field. In any case, the concern of this paper is only that there were available, for each subject, four films usually of varying quality.

The two observers who read the films twice gave about three-quarters of the films the same assessment of quality on the two occasions. In no case did either of these readers assess a film as good (+1) on one occasion and bad (−1) on another.

The most noticeable difference between the standards of the observers was that Reader C never gave a score of +1. Excluding this reader, there were seven readings of quality for each of the 344 films and all seven readings were in agreement for 87 (*i.e.* just over a quarter) of the films. There were only eight films where one observer gave an assessment of +1 and another an assessment of −1; no explanation for these discrepancies can be given.

The Data

For each of the 344 films there were thus eight assessments of quality (+1, 0 or −1) and eight readings of pneumoconiosis (0, 1, 2, 3, or P.M.F.). A "*Quality Score*" was obtained for each film by adding together the eight individual assessments. This procedure is not strictly justifiable (on statistical grounds) but because of the relative consistency of the assessors, it does provide fairly reliable differentiation between films, in terms of quality. A film which every assessor thought good would have a "Quality Score" of +8; one which every assessor thought bad, −8.

TABLE 2

SUMMARY OF ASSESSMENTS OF FILM QUALITY

Set	Reader A: 1st Occasion				Reader A: 2nd Occasion				Reader B: 1st Occasion				Reader B: 2nd Occasion			
	Scores			Total Score	Scores			Total Score	Scores			Total Score	Scores			Total Score
	−1	0	+1		−1	0	+1		−1	0	+1		−1	0	+1	
X2	16	35	35	+19	15	35	36	+21	11	48	27	+16	15	45	26	+11
X1	11	55	20	+9	9	50	27	+18	13	53	20	+7	12	54	20	+8
W	22	52	12	−10	14	59	13	−1	30	52	4	−26	33	50	3	−30
Y	34	46	6	−28	29	50	7	−22	33	52	1	−32	40	44	2	−38
	Reader C				Reader D				Reader E				Reader F			
	Scores			Total Score	Scores			Total Score	Scores			Total Score	Scores			Total Score
	−1	0	+1		−1	0	+1		−1	0	+1		−1	0	+1	
X2	33	53	0	−33	16	59	11	−5	17	64	5	−12	15	58	13	−2
X1	33	53	0	−33	11	63	12	+1	7	75	4	−3	11	58	17	+6
W	68	18	0	−68	32	53	1	−31	25	60	1	−24	20	51	15	−5
Y	62	24	0	−62	34	51	1	−33	24	61	1	−23	30	49	7	−23

TABLE 3

DISTRIBUTION OF FILMS BY "QUALITY SCORE"

"Quality Score" (Total of Eight Readings)	Number of Films		Classification
+8	0		
+7	2		
+6	6		
+5	12		
+4	16	154	"Acceptable"*
+3	19		
+2	23		
+1	25		
0	51		
−1	47		
−2	31		
−3	21		
−4	15		
−5	19	190	"Poor"
−6	17		
−7	19		
−8	21		
All	344		—

*For definition of "Acceptable" and "Poor" see text. This Table should not be taken to mean that more than half the films were unacceptable to *all* readers.

TABLE 4

DISTRIBUTION OF SUBJECTS BY "PNEUMOCONIOSIS SCORE"

"Pneumoconiosis Score" (Total of 32 Readings)	Group Title	Number of Subjects
0	"Pure normal"	13
1 to 8	"Lower O"	20
9 to 16	"Upper O"	9
17 to 48	"1"	16
49 to 80	"2"	11
81 to 96	"3"	3
Any one reading of P.M.F.	"P.M.F."	13
All	—	85*

*Excluding the one subject all of whose films were unreadable.

TABLE 5

DISTRIBUTION OF SUBJECTS ACCORDING TO THE NUMBERS OF "ACCEPTABLE" AND "POOR" FILMS

	Number of Subjects	
	Observed	Expected
All four films "Acceptable"	14	3·45
Three films "Acceptable", one "Poor"	13	17·05
Two films "Acceptable", two "Poor"	16	31·55
One film "Acceptable", three "Poor"	27	25·94
No films "Acceptable", all four "Poor"	16	8·00
Total	86	

The distribution of the films by "Quality Score" is given in Table 3, where it can be seen that roughly half the films had "Quality Scores" of 0 or better and these we defined as "*Acceptable*"; roughly half the films had scores of −1 or worse and these we defined as "*Poor*".

Similarly, a "*Pneumoconiosis Score*" was obtained for each subject by adding together the 32 readings (eight each for four films) for each subject,* unless any of the 32 readings was of P.M.F., in which case the subject was allocated to a "P.M.F." group. A "Pneumoconiosis Score" of O means that all 32 readings were normal; a "Pneumoconiosis Score" of 48 could mean that half the readings were category 1 and the other half category 2 so that the man's condition probably lies close to the borderline between these two categories. For a small number of subjects, a few readings were not available because particular observers had considered individual films "unreadable". Allowance has been made for these missing values to obtain an approximate "Pneumoconiosis Score". The distribution of the subjects by this measure is given in Table 4; in no case is the classification indicated in this Table likely to have been affected by "unreadable" films.

Consistency of Differences in Quality between Subjects

For each subject there are four films classifiable as "Acceptable" or "Poor". Table 5 gives the distribution of the 86 subjects according to the number of their films in these two classes. This Table also gives the expected distribution of the subjects on the assumption that the chance of any one film being classed as "Acceptable" or "Poor" is independent of the classification of any other film of the same subject. There is a clear tendency for many more subjects than expected to produce either four "Acceptable" films or four "Poor" films. This result is highly significant statistically: $\chi^2 = 48{\cdot}93$, with 3 degrees of freedom; $p << 0{\cdot}001$. Thus we conclude that the quality of a film depends to a considerable extent on the subject.

Factors Affecting Film Quality

We examined the relationship between film quality and the subject's age and chest thickness, for each pneumoconiosis group separately. In Table 6 we give for each group the average age, the average chest thickness, and the average "Quality Score". It is clear from this Table that the "Pure Normal" group differed markedly from those with evidence of pneumoconiosis, in that, on average, they were much younger, had thinner chests and considerably higher "Quality Scores". Amongst those with evidence of pneumoconiosis there was only a slight tendency for the men with more pneumoconiosis to be older than those with little

*Again this procedure is not strictly justifiable but it does provide fairly reliable differentiation between subjects, in terms of pneumoconiosis. An analogous procedure was used by Rivers, Wise, King, and Nagelschmidt (1960) who used a different scale of pneumoconiosis which should not be confused with the present "Pneumoconiosis Score" or the group titles defined in Table 4.

pneumoconiosis. There was no clear tendency for either chest thickness or "Quality Score" to be related to pneumoconiosis.

Within each pneumoconiosis group, we found a relationship between a subject's chest thickness and the average "Quality Score" of his four films. For men in the "Pure Normal" group the relationship was very slight. In the various groups of men with evidence of pneumoconiosis the relationships were similar: the average "Quality Score" decreased markedly with increasing chest thickness. One subject, excluded from these results because all of his films were assessed as "unreadable" by all readers, had a chest thickness of 32 cm., *i.e.* 3 cm. greater than any other subject. The results are presented in Fig. 1. It can be seen that the films for men in the "Pure Normal" group were generally assessed as better than for the men in the other groups, even when allowance has been made for chest thickness. There was no evidence from this examination that the age of the subject had any effect on the quality of his films.

Within each of the seven groups of subjects according to "Pneumoconiosis Score", the "Quality Scores" were related to the age and chest thickness of the subjects, for the films taken on each *x*-ray set separately. No association could be found with the age of the subject, but there was a general tendency for the "Quality Score" to decrease with increasing chest thickness. The effects differed from one group to another and for the different *x*-ray sets: they were hardly existent for the "Pure Normal" group; they were slightly more marked in the other groups for sets X1 and X2 than for sets W and Y. In general, the "Quality Scores" on sets X1 and X2 were markedly higher for men with comparatively thin chests, than those on sets W and Y, but for men with thick chests there was little difference in the quality of films from all four sets. For men in the "Pure Normal" group the assessment of quality was generally higher than for the other groups, when age and chest thickness had been allowed for. The results are presented diagrammatically in Fig. 2.

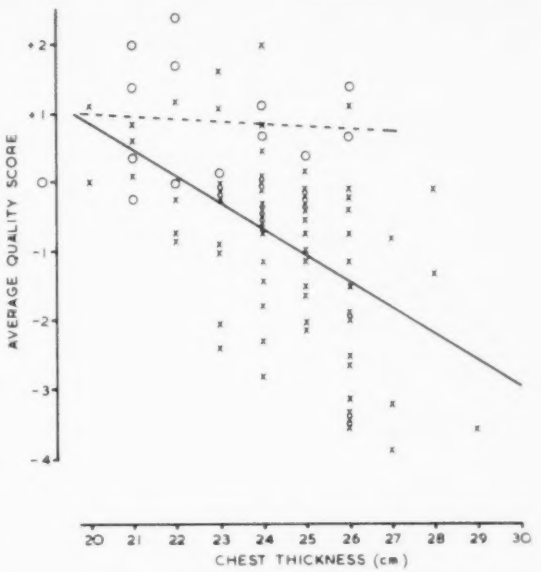

FIG. 1.—Chest thickness and film quality.
O Subjects in "Pure Normal" group — — — — —
X Other subjects ————

Factors Affecting Reading Error

Although Rivers *et al.* (1960) demonstrated an association between radiological category of simple pneumoconiosis and dust in the lung, it was by no means perfect. This is not unexpected because, as pointed out by Gilson (1960), the reading of a chest film is a descriptive process in that the classification of radiographs was originally developed on an empirical basis to provide a semi-quantitative scale of increasing abnormality. Indeed, any particular reading of a film is liable to variation from three causes. First, the film does not show the particles of dust themselves but only the opacities attributed to their retention in the lung; it may therefore be affected by the type of dust, by its distribution within the lung and by the radiological characteristics of the lung itself. Secondly, there is an element of observer error in assessing the number and size

TABLE 6

AVERAGE AGES, CHEST THICKNESSES, AND FILM "QUALITY SCORES" OF SUBJECTS IN PNEUMOCONIOSIS GROUPS

Pneumoconiosis Group	Number of Subjects	Average Age of Subjects (years)	Average Chest Thickness of Subjects (cm.)	Average "Quality Score" of Films of Subjects
"Pure normal"	13	34·5	22·9	+1·9
"Lower O"	20	45·4	24·4	−1·1
"Upper O"	9	54·0	24·8	−2·2
"1"	16	48·8	24·7	−1·9
"2 and 3"	14	48·9	23·9	−1·7
"P.M.F."	13	54·8	24·5	−2·1

TABLE 3

DISTRIBUTION OF FILMS BY "QUALITY SCORE"

"Quality Score" (Total of Eight Readings)	Number of Films		Classification
+8	0		
+7	2		
+6	6		
+5	12		
+4	16	154	"Acceptable"*
+3	19		
+2	23		
+1	25		
0	51		
−1	47		
−2	31		
−3	21		
−4	15		
−5	19	190	"Poor"
−6	17		
−7	19		
−8	21		
All	344		—

*For definition of "Acceptable" and "Poor" see text. This Table should not be taken to mean that more than half the films were unacceptable to *all* readers.

TABLE 4

DISTRIBUTION OF SUBJECTS BY "PNEUMOCONIOSIS SCORE"

"Pneumoconiosis Score" (Total of 32 Readings)	Group Title	Number of Subjects
0	"Pure normal"	13
1 to 8	"Lower O"	20
9 to 16	"Upper O"	9
17 to 48	"1"	16
49 to 80	"2"	11
81 to 96	"3"	3
Any one reading of P.M.F.	"P.M.F."	13
All	—	85*

*Excluding the one subject all of whose films were unreadable.

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The distribution of the films by "Quality Score" is given in Table 3, where it can be seen that roughly half the films had "Quality Scores" of 0 or better and these we defined as "*Acceptable*"; roughly half the films had scores of −1 or worse and these we defined as "*Poor*".

Similarly, a "*Pneumoconiosis Score*" was obtained for each subject by adding together the 32 readings (eight each for four films) for each subject,* unless any of the 32 readings was of P.M.F., in which case the subject was allocated to a "P.M.F." group. A "Pneumoconiosis Score" of O means that all 32 readings were normal; a "Pneumoconiosis Score" of 48 could mean that half the readings were category 1 and the other half category 2 so that the man's condition probably lies close to the borderline between these two categories. For a small number of subjects, a few readings were not available because particular observers had considered individual films "unreadable". Allowance has been made for these missing values to obtain an approximate "Pneumoconiosis Score". The distribution of the subjects by this measure is given in Table 4; in no case is the classification indicated in this Table likely to have been affected by "unreadable" films.

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Within each of the seven groups of subjects according to "Pneumoconiosis Score", the "Quality Scores" were related to the age and chest thickness of the subjects, for the films taken on each *x*-ray set separately. No association could be found with the age of the subject, but there was a general tendency for the "Quality Score" to decrease with increasing chest thickness. The effects differed from one group to another and for the different *x*-ray sets: they were hardly existent for the "Pure Normal" group; they were slightly more marked in the other groups for sets X1 and X2 than for sets W and Y. In general, the "Quality Scores" on sets X1 and X2 were markedly higher for men with comparatively thin chests, than those on sets W and Y, but for men with thick chests there was little difference in the quality of films from all four sets. For men in the "Pure Normal" group the assessment of quality was generally higher than for the other groups, when age and chest thickness had been allowed for. The results are presented diagrammatically in Fig. 2.

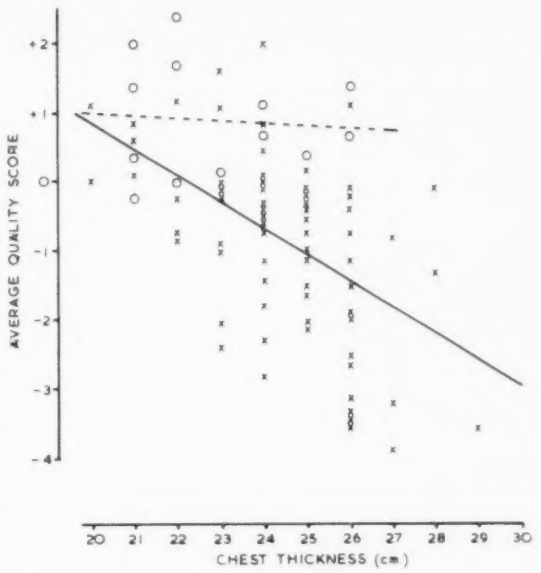

FIG. 1.—Chest thickness and film quality.
O Subjects in "Pure Normal" group — — — — —
X Other subjects ————

Factors Affecting Reading Error

Although Rivers *et al.* (1960) demonstrated an association between radiological category of simple pneumoconiosis and dust in the lung, it was by no means perfect. This is not unexpected because, as pointed out by Gilson (1960), the reading of a chest film is a descriptive process in that the classification of radiographs was originally developed on an empirical basis to provide a semi-quantitative scale of increasing abnormality. Indeed, any particular reading of a film is liable to variation from three causes. First, the film does not show the particles of dust themselves but only the opacities attributed to their retention in the lung; it may therefore be affected by the type of dust, by its distribution within the lung and by the radiological characteristics of the lung itself. Secondly, there is an element of observer error in assessing the number and size

TABLE 6

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Pneumoconiosis Group	Number of Subjects	Average Age of Subjects (years)	Average Chest Thickness of Subjects (cm.)	Average "Quality Score" of Films of Subjects
"Pure normal"	13	34·5	22·9	+1·9
"Lower O"	20	45·4	24·4	−1·1
"Upper O"	9	54·0	24·8	−2·2
"1"	16	48·8	24·7	−1·9
"2 and 3"	14	48·9	23·9	−1·7
"P.M.F."	13	54·8	24·5	−2·1

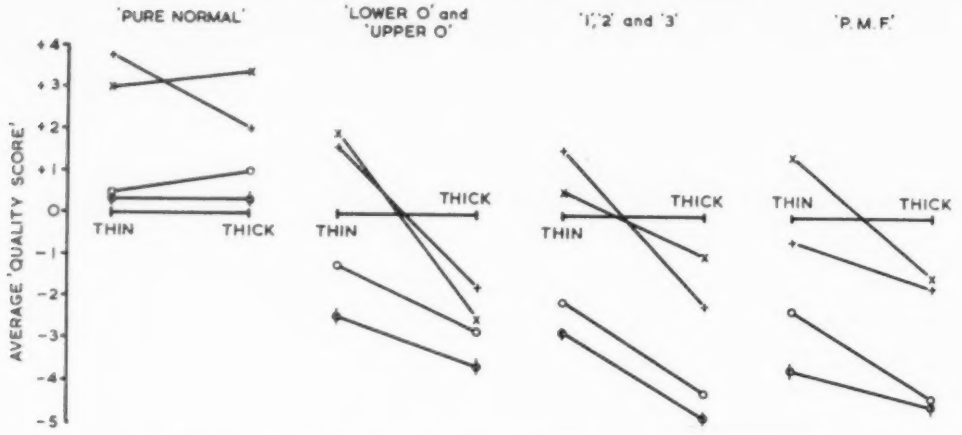

FIG. 2.—The relationship of film quality and *x*-ray set, chest thickness, and pneumoconiosis.
"Thin" = up to 24 cm. chest thickness; "Thick" = 25 cm. or more chest thickness.
X set X2 + set X1
O set W ϕ set Y

of the opacities in a particular film. Thirdly, radiographic technique affects the appearance of a film; readers unfamiliar with pneumoconiosis tend to see less of it in over-exposed or over-penetrated (*i.e.* black) films and more in under-exposed or under-penetrated (*i.e.* grey) films, although, of course, experienced readers make allowances for these effects.

The "accuracy" of film reading has often been studied in terms of the consistency with which a number of readers assess a batch of films or of the consistency with which one reader assesses a batch of films on separate occasions. The classical work of Fletcher and Oldham (1949) embraced the examination of the effects of technique. For each of nine subjects, they had available three films: one of acceptable technique; one made deliberately too black; one deliberately too grey. They found a general tendency for less abnormality to be read in the black films and more in the grey films, although some of their readers had made adequate (or even in some cases too great) allowances for technique.

The present data made it possible to extend this approach and to examine how successful readers are in making corrections for technique in the ordinary course of reading. It is also possible to assess whether bad technique increases observer error. These topics are discussed in the following two sections in terms of the readings of the films of the subjects who produced at least one acceptable film and at least one poor film (see Table 5). Amongst the 56 such men were 10 for whom P.M.F. was read and these have all been excluded from the present studies.

Effect of Film Quality on Reading Pneumoconiosis

To assess the effect of technique on reading, we compared, for each subject, the reading of the film which had the lowest "Quality Score" for that subject with the reading of that subject's film which had the highest "Quality Score". To ensure a reasonably marked difference in quality, we excluded those subjects where the difference between the "Quality Scores" on the best and worst films was less than four. We also excluded two subjects because readings were not available since particular readers considered at least one film "unreadable". For the remaining 30 subjects, the film with the worst "Quality Score" was re-examined to determine the reason for the low assessment of its quality. In 14 cases the worst film was too black; in nine it was too grey; in the remaining seven it was assessed as worst because of such factors as movement or incomplete inspiration.

The results are given in Table 7; this is in three parts, according to the reason for the worst film being so assessed. In each part of the Table, we give, for each subject separately, the "Quality Score" on the best and worst films and, for each of these two films, the eight individual readings of the films. On the right of the Table we give the average of the readings on the best and worst films, together with the difference.

From Table 7a, it can be seen that on average slightly more abnormality was read in films which were too black than in the best films of the same subject. The individual readers varied considerably; one had a general tendency to overread by about half a category on average whereas, at the other

extreme, another reader underread by about one third of a category. Again the tendency is by no means common to every subject; the worst films for subjects 77 and 41 were read by the majority of readers as having more abnormality than the best films of the same subject; for most other subjects on the other hand the differences between the best and worst films tended to cancel out or were in any case infrequent.

From Table 7b, it can be seen that on average slightly less abnormality was read in the films which were too grey than in the best films of the same

TABLE 7a

READINGS ON BEST AND WORST FILMS: 14 SUBJECTS WHOSE WORST FILMS WERE BLACK

Subject		77		41		44		74		7		30		1		40		42		11		24		5		9		6		Average For All 14 Subjects		
Set "Quality Score"		X2 0	Y −5	X2 0	Y −7	X2 +2	W −4	X1 +3	Y −4	X2 +6	Y −5	X2 +2	W −6	X1 0	W −8	X2 +3	X1 −5	X1 0	Y −8	X1 +2	W −4	X1 0	Y −5	X1 0	Y −7	X2 +4	W −6	X2 +6	Y −5	Best Films	Worst Films	Difference
Reader*	E	0	0	1	2	0	0	1	3	0	1	0	1	2	2	0	0	0	1	1	2	0	0	1	0	0	0	2	2	0·57	1·00	+0·43
	A1	0	1	1	2	1	2	2	2	0	0	1	1	1	1	0	0	1	1	2	2	0	0	1	1	0	0	2	2	0·86	1·07	+0·21
	A2	0	2	1	2	1	2	2	2	0	0	1	1	1	1	0	0	1	1	2	2	0	0	1	1	1	0	3	2	1·00	1·14	+0·14
	B1	1	2	1	2	1	2	2	2	0	0	1	1	1	1	0	0	1	0	2	2	0	0	1	1	0	0	2	1	0·93	1·00	+0·07
	B2	0	2	1	2	1	2	2	2	0	0	1	0	1	1	0	0	1	1	2	2	0	0	0	0	0	0	2	1	0·79	0·86	+0·07
	C	1	2	2	2	1	2	3	3	0	0	1	2	2	2	0	0	1	0	2	1	0	0	2	2	1	0	1	2	1·21	1·29	+0·08
	D	1	1	1	1	1	2	2	2	0	0	1	1	1	1	0	0	1	1	2	2	1	0	1	1	0	1	2	2	1·07	1·00	−0·07
	F	1	1	1	2	2	1	2	2	0	0	0	0	2	1	0	0	2	2	3	2	0	0	1	0	1	0	3	2	1·29	0·93	−0·36
Difference of average of all readings		+0·88		+0·75		+0·38		+0·25		+0·12		+0·12		−0·12		−0·12		−0·12		−0·12		−0·12		−0·25		−0·25		−0·38		—		+0·07

TABLE 7b

READINGS ON BEST AND WORST FILMS: NINE SUBJECTS WHOSE WORST FILMS WERE GREY

Subject		62		78		53		82		33		49		45		58		69		Average for All Nine Subjects		
Set "Quality Score"		X2 0	Y −7	X2 +2	Y −5	X1 0	X2 −4	X2 +1	Y −4	X2 +1	W −4	X2 +1	W −7	X1 +1	X2 −7	X1 0	X2 −8	X2 0	Y −8	Best Films	Worst Films	Difference
Reader*	A1	1	1	0	0	0	0	1	0	1	1	0	0	0	0	1	0	2	1	0·67	0·33	−0·34
	A2	2	2	0	0	0	0	1	1	1	1	0	0	0	0	1	0	3	2	0·89	0·67	−0·22
	D	1	1	0	0	1	0	1	1	1	1	0	0	0	0	1	1	2	2	0·78	0·67	−0·11
	B1	2	2	0	0	1	0	0	0	1	1	0	0	0	0	0	1	3	3	0·78	0·78	0
	B2	2	1	0	0	0	0	0	0	1	1	0	0	0	0	0	0	2	3	0·56	0·56	0
	F	0	1	0	0	0	2	0	0	2	2	0	0	1	0	1	0	2	1	0·67	0·67	0
	C	2	2	0	1	1	1	1	2	2	2	1	0	0	0	1	1	3	2	1·22	1·22	0
	E	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	1	0·22	0·33	+0·11
Difference of average of all readings		+0·25		+0·12		0		0		0		−0·12		−0·12		−0·25		−0·50		—		−0·07

TABLE 7c

READINGS ON BEST AND WORST FILMS: SEVEN SUBJECTS WHOSE WORST FILMS WERE SO ASSESSED FOR OTHER REASONS

Subject		37		80		64		29		52		34		28		Average for All Seven Subjects		
Set "Quality Score"		X2 +2	W −3	X2 +5	Y −2	X1 +4	Y −1	X2 +5	Y −1	X1 +2	X2 −2	X2 +3	Y −2	X2 +5	W −3	Best Films	Worst Films	Difference
Reader*	C	1	2	1	1	2	2	0	1	0	1	1	0	3	3	1·14	1·43	+0·29
	B1	0	1	0	1	2	2	0	0	0	0	0	0	2	2	0·57	0·86	+0·29
	F	0	2	0	0	2	3	0	0	1	0	1	0	3	3	1·00	1·14	+0·14
	A2	0	1	0	0	2	2	0	0	0	0	1	1	3	3	0·86	1·00	+0·14
	E	0	0	0	0	1	2	0	0	0	0	0	0	3	2	0·57	0·57	0
	A1	1	1	0	0	2	1	0	0	0	0	1	1	3	3	1·00	0·86	−0·14
	B2	0	0	0	0	2	2	0	0	0	0	0	0	3	2	0·71	0·57	−0·14
	D	1	1	1	1	1	1	1	0	1	0	1	1	3	2	1·29	0·86	−0·43
Difference of average of all readings		+0·62		+0·12		+0·12		0		−0·12		−0·25		−0·38		—		+0·01

*For Readers A and B, the digit indicates the occasion on which the films were read.
For each subject readings on the best films are on the left.

TABLE 8

DISCREPANCIES BETWEEN READING: A, THE BEST AND NEXT BEST FILM FOR EACH SUBJECT; B, THE BEST AND WORST FILM FOR EACH SUBJECT

Subject	Sets and "Quality Scores"			A: Readers Recording Differences Between Best and Next Best Films	Total	B: Readers Recording Differences Between Best and Worst Films	Total
	Best	Next Best	Worst				
7	X2(+6)	X1(+4)	Y(−5)		0	E	1
6	X2(+6)	X1(+3)	Y(−5)	C	1	A2 B1 B2 C F	5
9	X2(+4)	X1(+3)	W(−6)	A2 C D F	4	A2 C D F	4
74	X1(+3)	X2(+1)	Y(−4)	A1 C D E	4	A1 C D E*	4*
4	X2(+4)	X1(+2)	W(−3)		0		0
80	X2(+5)	X1(+3)	Y(−2)		0		0
29	X2(+5)	X1(+3)	Y(−1)		0	C D	2
44	X2(+2)	X1(+1)	W(−4)	A1 A2 B1 B2 D E	6	A1 A2 B1 C F	5
64	X1(+4)	W(+3)	Y(−1)	A1 A2 B1 B2 C D E F	8	A1 E F	3
33	X2(+1)	X1 (0)	Y(−3)	C F	2	B2 E F*	3*
17	X1(+3)	X2(+1)	W(−1)		0		0

*Discrepancy of two categories.

subject. Again there were differences between readers and between subjects.

As might be expected, little pattern emerges from an examination of Table 7c.

Effect of Film Technique on Repeatability of Reading

In the previous section we tried to discover whether film quality introduced a bias into readings. In this section we consider whether the reading of films of poor technique shows greater variation than the reading of films of good technique. We used two methods.

In the first method we compared the numbers of discrepancies in reading pairs of films of the same subject (a) when the films were both "Acceptable" (and their "Quality Scores" close together) and (b) when one film was "Acceptable" and the other "Poor" (and their "Quality Scores" were reasonably far apart). In Table 8 we list the 11 subjects whose films met the following criteria:

(i) At least two were "Acceptable" and at least one "Poor" film;

(ii) The "Quality Score" on the best film was at least four points greater than the "Quality Score" on the worst film;

(iii) The "Quality Score" on the second best film was within three points of the "Quality Score" on the best film.

A small number of subjects was excluded because two films had equal "Quality Scores".

In Table 8 we also indicate those readers whose readings for these 11 subjects differed (a) when reading the best and next best films and (b) when reading the best and worst films. An asterisk indicates that the difference was of two categories, otherwise all differences were of one category only. For five subjects there were more discrepancies when comparing best and worst films than when comparing best and next best films; for two subjects there were fewer discrepancies and for the remaining six subjects there were equal numbers. Similarly, two readers showed more differences between best and worst films than between best and next best films; with one reader this tendency was reversed but with the others there were no differences.

In the second method of comparison, we used the material of Table 7 for readers A and B, both of whom read the films twice. One reader had slightly greater variability on the worst films than he had on the best, but for the other reader the tendency was reversed. In neither case was the difference in variability significant.

Discussion

It is not easy to quantify the results, especially as they related to six particular readers, whose general levels of reading pneumoconiosis differed considerably. However, for these six, differences in film quality appeared to introduce about the same lack of repeatability as did differences between readers with similar levels. In the extreme, the two readers whose levels differed most had many more disagreements with each other in their readings of the best films than each had, separately, between his readings of the best and worst films.

These findings are not obviously in line with those from the only other work on this subject known to the author: that of Fletcher and Oldham (1949) and that, as yet unpublished, of Wise (1959). However, comparison with either is difficult. Fletcher and Oldham only used nine subjects, and the variations in technique were deliberately introduced by altering the factors on one set. The changes produced may not be strictly comparable with the variations produced by the different sets in the present trial.

There is, however, one important difference that does seem valid. The earlier work showed that the errors were, on average, in the "expected" direction (*i.e.* more abnormality was read in grey films, less in

black) whereas in the current study these tendencies were, if anything, reversed.

The material examined by Wise consisted of only one film for each subject. He assumed that the real distribution of abnormality (*i.e.* what ought to be read on perfect films) would be independent of film technique. The current study suggests that this assumption is not tenable.

Three further considerations must be borne in mind. First, the present sample of films was heavily biased by the exclusion of men without pneumoconiosis. Secondly, it was also biased by the inclusion of a high proportion of men whose earlier (1955) films had been read differently on two occasions by one experienced reader; difficulty in reading was thus not unexpected. Thirdly, since the films which were easiest to read were generally assessed as of good quality, there may be an important source of inaccuracy in the assessment of quality, which is clearly a subjective process.

Conclusions

(a) The quality of a film depends on the subject; in the circumstances of this trial, which simulated survey conditions, chest thickness but not age had an important effect on the average quality of the films. Also those films showing pneumoconiosis were, for equal chest thickness, given a lower "quality" grading than those without pneumoconiosis.

(b) For those 30 subjects who had both good and bad films, film technique did not appear to introduce consistent biases into film reading nor any greater variability. In other words, the particular readers were in general able to make reasonable allowances for the effects of technique on the film's appearance.

(c) There are subjects, especially those with thick chests, in whom it is difficult to get technically good films, and it is in these that the effects of poor technique probably affect the accuracy of reading of pneumoconiosis most.

I should like to thank all those who took part in the trial and, in particular, Dr. J. C. Gilson. I owe a special debt of gratitude to Dr. P. J. Chapman, who conducted the trial and with whom I have had many very helpful discussions.

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APPENDIX

Radiographic Details

Before the trial was undertaken preliminary meetings were held with all the manufacturers where it was agreed that the target film distance would be standardized at 60 in. and that no added filtration (except that built into the tubes) would be used. It was also agreed that the maximum focal spot size would be 2 mm., but all manufacturers used tubes with nominal focal spot sizes 1·2 mm.

The tubes were all virtually new.

For use with the one method of exposure control, three cassettes were selected at random from amongst those belonging to the National Coal Board No. 2 Research *X*-ray Unit. A special type of cassette was necessary for use with the other method and such cassettes were provided by the manufacturer.

The intensifying screens, films, and chemicals were provided by the National Coal Board. High definition screens (as used by the N.C.B. Research *X*-ray Units) were provided and before the trial six pairs of screens were tested for comparability of speed and definition. The "speed" of the screen was compared using an aluminium step wedge and the definition compared using metal studding (threaded rod) of various sizes. Visual comparison showed no detectable difference between pairs of screens. Three pairs of screens, chosen at random, were mounted in the N.C.B. Research Unit's cassettes and the other three pairs of screens were mounted in the special cassettes for use with the second method of exposure control.*

The films used were 14 in. × 14 in. Kodak Standard Series 6, all of one emulsion mix. Sheets of film were taken at random from each box of 75 films and tested for comparability using similar methods to those employed in testing the screens. The *x*-ray equipment used for these tests was that of the N.C.B.'s Unit (Siemens Neo-Heliophos with four-valve full-wave rectification).

Darkroom, developing facilities, and chemicals were provided by the N.C.B. *X*-ray Unit. All films were handled and developed in the one darkroom by the same N.C.B. technician throughout. The developer used was Kodak DX.80, and the fixer Kodak *x*-ray fixer with hardener. Development time was standardized (five minutes at 68° F.) for all films, using an automatic (Hills Russell) unit. A graphic record of the temperature of the developer was maintained by a recording thermograph, and showed that the temperature during each developing session was equal and constant. At no time did the temperature vary by more than ± 1° F.

The developer was replenished at intervals, the amount

*See also last paragraph of this Appendix.

added depending on the total area of exposed films processed (29 fl. oz., per 27 sq. ft.). In practice this means 29 fl. oz., for each 20 full-size radiographs developed.

The developing conditions throughout the trial were assessed by special test films. On each of these test strips, made from Kodak Standard Series 6 *x*-ray film, there was a density step wedge prepared by exposure to a constant low wattage light source. The test films were processed at intervals and the densities on corresponding portions of different test films were measured with a Baldwin Vacuum Cell densitometer. By this means any gross variations in the developer likely to affect the finished *x*-ray films would be detected. Once testing was complete and the trial had started there was virtually no change in the shapes of the curves relating density to log exposure and no variation in the developer appears sufficient to affect the finished radiograph.

In addition to those men selected for the trial proper, 80 had radiographs taken by the N.C.B. *X*-ray Unit before the trial started and the exposed films were processed in the solutions to be used for the rest of the trial; thus the initial comparatively rapid changes in the developing solutions took place before the trial films were processed.

The details of radiographic techniques were left to the discretion of the individual operators within the broad limits that at least 300 mA should be employed and that the range of kv should be that conventionally used for full-size chest radiography.

To enable the radiographers in charge of the three *x*-ray sets to decide the details of their radiographic technique a selection was made of a further 20 men. (With the 100 men chosen for the trial proper and the 80 mentioned above, a total of 200 was involved in the trial.) These men, 10 of whom attended on each of two days, represented a range of age, chest thickness, and radiological appearances. Their *x*-ray films were handled and processed under the conditions of the trial.

When each man presented, he was identified and allotted a card containing an identifying number punched into a leaded strip. He was also allocated (in rotation) one cassette of each type. One of the N.C.B. research team escorted the man, ensuring that he was *x*-rayed on each set and that the number on the leaded card was entered, by *x*-radiation, on each of his films. The same cassette and thus the same pair of screens was used for all the one man's films taken with the exposure control common to those sets. Thus on each of the four sets, three cassettes were used in rotation.

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A SURVEY OF THE METHODS DEVELOPED IN THE NATIONAL COAL BOARD'S PNEUMOCONIOSIS FIELD RESEARCH FOR CORRELATING ENVIRONMENTAL EXPOSURE WITH MEDICAL CONDITION

BY

J. W. J. FAY and J. R. ASHFORD

From the Pneumoconiosis Field Research Unit, National Coal Board, London

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The correlation of the medical and environmental data (*i.e.* the derivation of the dosage–response relationship) in a study such as the National Coal Board's Pneumoconiosis Field Research (P.F.R.) is subject to many complicating factors compared with the more conventional types of biological assay. Several methods have been developed within the Research to overcome these difficulties, and the new procedures are described. Each is concerned with the estimation of the direct relation between the radiological abnormality associated with simple pneumoconiosis and some single measure of the past hazard, but the basic techniques are sufficiently general to be applicable in other fields of study.

The first development involves the definition of an underlying continuous scale of radiological abnormality. This prepares the way for the derivation of the "quantitative" relation between exposure and response, to replace the "semi-quantal" relation which is inherent in the use of a small number of discrete categories of radiological abnormality. The effect of errors of observation of dosage and response on the corresponding quantitative and quantal relationships is then determined.

The second development concerns the use of a "multi-dimensional" representation of past hazard. Most of the men under observation had worked in a number of different mining occupations before their first chest radiograph was taken, but this exposure cannot be assessed in terms of dust concentrations, for which reliable data are not available. Nevertheless, it is shown that past hazard can usefully be represented by three "dimensions" corresponding to the periods spent in three main types of environment—(a) the coal-face (coal-getting shift), (b) the coal-face (preparation shift) and (c) elsewhere underground. Each man's past exposure up to the time of his first chest radiograph can be expressed in terms of these three dimensions and the effect of each environment separately can be determined.

The third development extends the multi-dimensional approach to cover not only the working history before the first medical examination, but also the recorded exposure (in terms of measured dust concentrations), to which each man has been subject between the first and subsequent "follow-up" surveys. This measured exposure is regarded as one dimension of the man's total exposure up to the time of his second (or later) examination, and it is possible in this way to determine the direct relation between radiological abnormality and measured exposure, even when this component represents only part of the total hazard to which the man has been subject.

The application of the methods is illustrated by the analysis of some of the data which have been obtained in the Pneumoconiosis Field Research.

One of the main objectives of the National Coal Board's Pneumoconiosis Field Research (P.F.R.) is to determine what environmental conditions should be maintained in British collieries if the miners are not to be disabled by the dust they breathe in the course of their work. The problem is

being tackled by a study of the relation between the prevalence and progression of pneumoconiosis and the environmental conditions, particularly airborne dust concentrations, at a number of collieries located throughout the coalfields in the United Kingdom (Fay, 1957).

The population under examination comprises the underground and surface workers at 25 selected collieries, amounting to about 35,000 men in all. A series of radiological surveys is being made at each colliery at intervals of about four years. Since 1953, when the Research was started, a continuous programme of environmental measurement has been carried out at each colliery, thus providing a record of dust exposure to match against radiological change for each man (Fay and Rae, 1959).

The correlation of the resulting medical and environmental data presents a task of some magnitude and complexity. Dosage–response relationships in biological assay work are commonly expressed in terms of the probability of a single "yes – no" (quantal) response to a known stimulus, *e.g.* the death of a test animal as the result of the controlled administration of a given dose of a single poison, but this simple situation is not present in a study such as the Pneumoconiosis Field Research. For one thing, the data are "uncontrolled", in the sense that the observer cannot determine the "doses" applied, having instead to make the best use he can of whatever information he is able to derive from his study of the subjects under examination. The use of such "uncontrolled" data is, of course, by no means a new development; Bull and Squire (1949), for example, applied the method of probit analysis to the study of mortality in relation to area of burns. In the present investigation the position is further complicated by a number of important factors.

The subjects are under observation with respect to both radiological pneumoconiosis and dust exposure. The "response" is measured, not in terms of a simple "dead or alive" observation, but in terms of more than one discrete stage of severity, as represented by the various radiological categories of simple pneumoconiosis. An essentially continuous process, whereby increasing amounts of dust produce increasing amounts of abnormality, is thus restricted to expression in quantal terms, using the steps corresponding to the discrete categories of pneumoconiosis. Obviously it is desirable to remove this limitation, and to establish a quantitative relation between exposure and response. This involves the derivation of the underlying continuous scale of radiological abnormality, and this particular step is the first main development described in this paper. It is shown that by the choice of a suitable scale of abnormality a straight-line relation can be obtained between degree of pneumoconiosis (measured on a continuous scale) and a logarithmic function of environmental exposure. This quantitative description represents a generalization of the quantal relationships governing the separate responses in terms of category 1 or more or category 2 or more pneumoconiosis, and provides a better insight into the processes involved than the simpler quantal response technique based on the discrete category boundaries implicit in the I.L.O. scale (International Labour Organization, 1953). The application of the method is illustrated by means of data obtained on the initial medical survey at one of the Pneumoconiosis Field Research selected collieries for a group of men who had worked only in one particular occupation.

In studies such as this the estimates of radiological abnormality and past hazard used for the dosage–response analysis may be subject to considerable errors of measurement. The effect of these errors on the quantitative abnormality–exposure relation is described and it is shown how the corresponding quantal relationship is modified by the uncertainties in the data.

The third major difficulty in the present study arises from the fact that the "dose" causing the response is not usually capable of expression in terms of a single parameter, because of the absence of any reliable quantitative information about exposure before the first medical surveys. It is, however, possible to obtain a measure of a man's past exposure from his working history, in terms of the periods spent in various types of environment. The majority of the men worked in more than one environment during the period before the initial medical surveys, and were thereby exposed to more than one level of hazard. It is therefore necessary to take account of a number of different "dimensions" of past exposure. A method of analysis has been developed to determine the dosage–response relationship associated with each dimension separately on the basis of the information from men with mixed past working histories. The application of this approach is illustrated by an analysis of the past exposure histories obtained from the whole population examined on the initial radiological survey at one of the Pneumoconiosis Field Research collieries. The advantages of this development will be immediately apparent, in that use is made of the data available for all subjects, instead of the analysis having to be restricted to the proportion (often small) of subjects who have been exposed to only one level of the hazard.

The information about past exposures up to the

time of the first medical surveys is limited to the periods spent in different types of environment, as described above. Since then, however, an extensive programme of airborne dust sampling and measurement has been followed, and during the four years or so between the first and second radiological examinations each man's dust exposure has been continuously recorded in quantitative terms. Hence, a man's cumulative exposure since joining the coal-mining industry can be regarded as a "multi-dimensional" variable, in terms of his different types of exposure before the first medical survey, together with one or more additional "dimensions" corresponding to his measured exposure between the first and second medical surveys. The use of the multi-dimensional method has therefore been extended to the analysis of the "progression" data, obtained from the follow-up medical surveys. In this way the probability that a man has developed a given category of simple pneumoconiosis or more at the time of (a) the first and (b) the second medical survey may be expressed as a function of the two sets of multi-dimensional variables, and the effect of the measured component alone may be assessed. The method has been developed to cover alternative procedures for reading the chest radiographs. Its use to estimate the relation between radiological abnormality and measured exposure is illustrated by means of the analysis of the data from the first and second radiological surveys at one of the Pneumoconiosis Field Research collieries. It must be emphasized that the figures quoted are used at this stage purely for the purposes of illustration of the method, and no importance should be attached in a quantitative sense to the results deduced.

In the following sections of this paper a general description is given of these various new methods, which are applicable in other fields than the study of coalworkers' pneumoconiosis. Full details of the underlying mathematical treatments have been published elsewhere (Ashford, 1958a; Ashford, 1959; Ashford, Smith, and Brown, 1960).

Medical Data

On each medical survey a 14 in. × 14 in. postero-anterior (P.A.) chest radiograph is taken of every man examined, and the readings of these films are the source of information about the extent of the man's radiological pneumoconiosis. On the second and subsequent rounds of medical surveys supplementary measurements are also made of lung function, anthropometry, and respiratory symptoms (Rogan, Ashford, Chapman, Duffield, Fay, and Rae, 1961). The main application of the new methods of correlation is, however, in terms of the radiological readings, and the present paper is concerned only with this aspect, so far as the medical data are concerned.

The categories of pneumoconiosis which have been used in the medical surveys are those accepted by international agreement (International Labour Organization, 1953), which recognizes a distinction between simple and complicated pneumoconiosis [or progressive massive fibrosis (P.M.F.)]. Simple pneumoconiosis is subdivided on a quantitative basis into three categories, increasing in degree of abnormality and numbered 1 to 3, category 0 being reserved for films which are "within normal limits". Films showing evidence of complicated pneumoconiosis are classified separately into four categories–A, B, C, and D. Because of the comparatively small number of men with P.M.F., and the uncertainties about the mechanism by which this condition becomes superimposed on simple pneumoconiosis (Gough, 1947; Cochrane, Cox, and Jarman, 1952), the methods of correlation described below are confined to the development of simple pneumoconiosis.

In view of the considerable element of doubt associated with a single reading of pneumoconiosis, even by experienced observers (Fletcher and Oldham, 1949), the final or "definitive" reading on each film is based on a series of readings by two doctors. The procedures applied and the results achieved have been described in detail elsewhere (Fay and Ashford, 1960; Ashford, 1960). Essentially, it has been the standard practice for each film to be read independently by the two doctors. Films on which these "routine" readings are not consistent are subsequently read again by both doctors in consultation and the definitive category is assigned as the result of their joint discussion. The readings of the films obtained on the second round of surveys have so far been made without reference to the corresponding first survey films, although consideration is currently being given to the possible application of other methods of reading. To check that the standards of reading on successive surveys at each colliery are comparable, a sample of the first survey films is read again at the time of the second survey. All the routine and definitive readings are made without reference to the man's past environmental history.

Although great care is exercised in taking and processing radiographs in order to maintain a consistent standard of radiographic technique, there are inevitably some variations in the quality of the films obtained on the various surveys. Superimposed upon these variations in producing the radiographs are the errors associated with the film reading process.

Environmental Data

In view of the absence of sufficiently reliable environmental measurements at the selected collieries before the beginning of the Research, no quantitative information is available about the man's previous exposure to dust. However, at the time of the first medical survey a working history was obtained of each man presenting himself for examination, showing the time spent in each of his various occupations and working places since leaving school. This information was checked against an independently prepared history of the colliery and subsequently summarized by the use of an electronic computer (Hicks, Fay, Ashford, and Rae, 1961) in terms of the periods spent in each of the six main types of general environment [hard heading, coal-face (coal-getting shift), coal-face (preparation shift), elsewhere underground, surface "dusty" and surface "non-dusty"] at the colliery in question and at other collieries. Each man is also assigned to a "pit" classification and "exposure" category on the basis of his past record, so that groups of men who have consistently followed the same occupation at the same colliery can be identified. These methods are, of course, an extension of the classical work done in South Wales by the Medical Research Council (Hart and Aslett, 1942). The working histories provide the only reliable information about the environmental exposures of the men before the first medical survey. This component will represent an appreciable part of the total exposure of the majority of the population for many years to come, and one of the main incentives for the development of new methods of correlation was the need to make the best possible use of these summarized past histories.

A systematic programme of environmental measurement has been carried out at each of the selected collieries since the Research started and the results obtained provide a basis for reliable estimates of individual exposures from the time of the initial medical surveys. The sampling procedure is based on an extension of the "random collier" method of Patterson (1939) and Oldham and Roach (1952). At each colliery the underground and surface populations are subdivided into a number of homogeneous strata, or occupational groups, consisting of men working at the same job, at the same place, at the same time. In this way the environment within each group may be expected to be reasonably uniform, and this is, of course, automatically checked by the subsequent measurements. By sampling a series of randomly selected individual workmen throughout a complete shift, the "exposure index" corresponding to each occupational group is determined, and this figure is attributed to each of the men belonging to the group, for the appropriate period which he spent in that group. Subsequent reference to the detailed record which is maintained of the attendance and movement from group to group of all men on the colliery register enables the individual environmental exposures to be calculated for any period of time after the first medical survey.

The dust concentrations are measured with the thermal precipitator and expressed in terms of the number of particles in the "respirable" size range per unit volume of air. The standard count is made in the 1-5 micron size range, and the relative proportions of "coal" and "non-coal" (*i.e.* mineral) are assessed. In addition, size distribution analyses are made on a proportion of the samples, in terms of the particle size ranges 0·5-1μ, 1-2·5μ, 2·5-5μ, and above 5μ. These measurements are supplemented by compositional analyses of the respirable dust, using microscopic, *x*-ray diffraction, and chemical methods, and on this basis a considerable variety of alternative measures of exposure is available for investigation.

In summary, the total environmental exposure of each individual since joining the mining industry can be subdivided into two distinct components, representing respectively his working history up to the time of the first medical survey, expressed in terms of periods in different environments, and his subsequent exposure, based on measured dust concentrations and time at risk, expressed in terms of any one of a number of different parameters. When applied as a measure of environmental exposure the working histories may be regarded as subject to errors of measurement, due to both vagaries of memory and variations in the hazard corresponding to the same class of environment. The measured component of exposure is also subject to errors of estimation, which are controlled by systematic adjustment of the allocation of sampling shifts between the occupational groups (Ashford, 1958b).

Relation between Prevalence of Simple Pneumoconiosis and Environmental Exposure

The Underlying Relationship.—It is generally accepted that simple pneumoconiosis is caused by the inhalation and retention of airborne dust in the lungs. Although the exact way in which the process takes place is not clearly understood at the present time, it is reasonable to suppose that the characteristic radiological abnormalities will be related to the quantity and composition of the dust retained and/or any corresponding tissue reaction (Rivers, Wise, King, and Nagelschmidt, 1960). In coalworkers' pneumoconiosis the situation is complicated by the possible intervention of progressive massive fibrosis

(P.M.F.), a condition which may be due to the superimposition of a tubercular infection upon a background of fairly advanced simple pneumoconiosis (Gough, 1947; Cochrane *et al.*, 1952; Cochrane and Miall, 1956). It has been shown (Stewart, 1948; Cochrane, 1960) that P.M.F. may progress after dust exposure has ceased. In simple pneumoconiosis, however, it is believed that this does not occur and that the development of the condition can be regarded as a continuous process, the amount of abnormality revealed in the radiograph increasing with increasing amounts of dust retained in the lungs.

Unfortunately, it is not possible to obtain a precise measure of this continuous variation in radiological abnormality, in view of the inherent difficulties of reading the radiographs. The best that can be done is to subdivide the abnormality scale in terms of a number of discrete categories; the I.L.O. classification used in these surveys divides the continuum of abnormality into four intervals, denoted respectively categories 0, 1, 2, and 3. As the abnormality value increases, the position of the film moves along the scale from the lower limit of category 0 to the upper, then over the boundary between categories 0 and 1, across category 1 and so on, until the upper limit of category 3 is reached or P.M.F. intervenes. Thus any given category of simple pneumoconiosis covers a range of abnormality values; in particular, the upper part of category 0 includes some films with radiological abnormality just insufficient to attain the category 1 classification.

We have seen that if a given individual is exposed to airborne dust his radiological abnormality will, in the absence of P.M.F., tend to increase with increasing exposure and, starting from the lower limit of category 0, will traverse each of the categories of simple pneumoconiosis in turn. The precise form of this abnormality–exposure relation will depend on the way in which these two quantities are measured, *i.e.* on the choice of the abnormality and exposure scales. From the point of view of any mathematical analysis the most convenient form of relationship is a straight line, and it is therefore desirable to choose the scales accordingly.

The classification of simple pneumoconiosis into categories 0, 1, 2, and 3 is based on an arbitrary subdivision of the continuum of abnormality, and the category numbers have no arithmetical significance in this context. In other words, there is no reason to suppose that each category covers an equal part of the abnormality scale or that category 0 represents abnormality values between 0 and 1 on the scale, category 1 represents abnormality values between 1 and 2, and so on. The absence of any basic measure of abnormality means that we are free to choose any convenient scale. It can be shown mathematically that a scale which leads to a straight line abnormality–exposure relation always exists, provided only that abnormality increases with increasing exposure. When the abnormality scale is defined the position of the origin and the scaling factor are arbitrary, and in the analyses to be described the boundary between categories 0 and 1 is taken as zero on the scale, and the boundary between categories 1 and 2 as unity.

The choice of a suitable scale for the exposure variable is a more straightforward matter. As in many other similar applications (Finney, 1952) it is most convenient to work in terms of the logarithm of the exposure variable, denoted the "dosage". Thus, for any given individual, the abnormality–exposure relationship takes the form shown in Fig. 1.

It follows that the negative abnormality values correspond to category 0, the values between 0 and 1 correspond to category 1, and the values in excess of 1 correspond to category 2, followed by category 3. Two of the category boundaries are arbitrary and the third boundary—between categories 2 and 3—can only be determined by reference to the natural history of pneumoconiosis amongst human populations. The development of simple pneumoconiosis beyond a reasonably advanced stage (generally regarded as about the lower limit of category 2) may be complicated by the superimposition of P.M.F. (Cochrane, Davies, Chapman, and Rae, 1956). As this possibility cannot be excluded amongst the populations under examination it is not possible to determine the precise position of the boundary between categories 2 and 3 on the scale. From the point of view of the subsequent analysis this is not important, as the calculations are concerned only with the boundaries between categories 0 and 1 and between categories 1 and 2.

For the particular individual in question, radiological abnormality (y_o) and dosage (x_e) are assumed to obey a linear relation of the form,

$$y_o = \alpha_o + \beta_o x_o, \qquad (1)$$

where α_o and β_o are two parameters which characterize his response to the particular measure of exposure under consideration. In this context the parameter β_o is necessarily positive, as it is assumed that abnormality must increase with increasing exposure.

When a population of subjects is exposed to a given hazard the corresponding individual responses will not be identical, because of the differences from man to man which exist in human populations. It is assumed that these individual variations may be

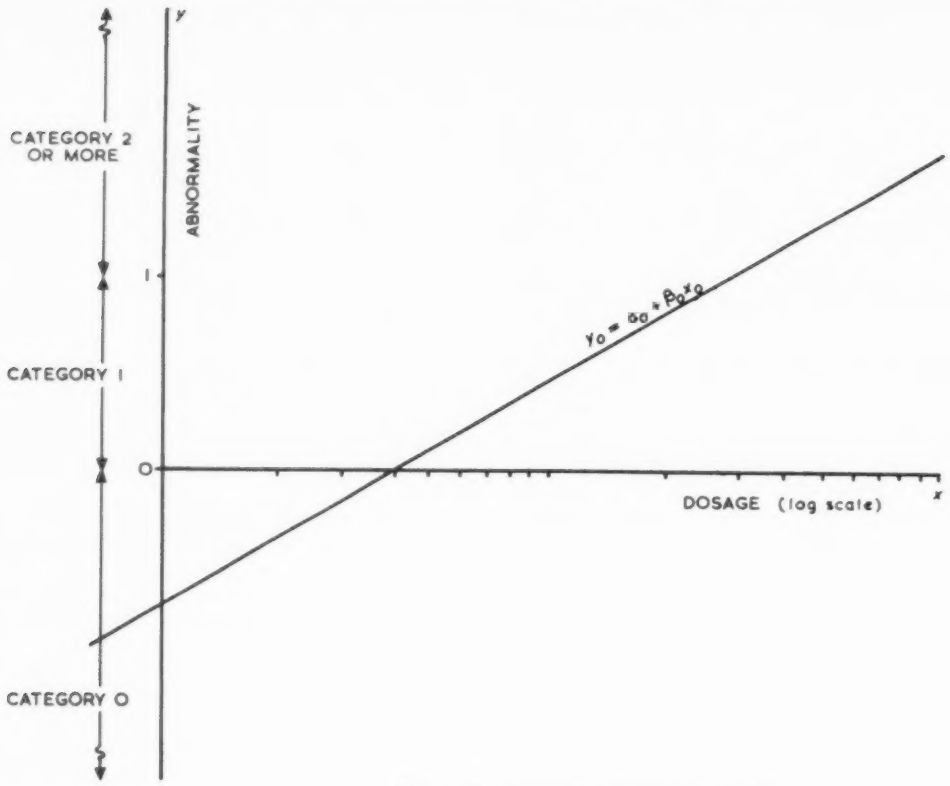

FIG. 1.—The abnormality–exposure relationship.

represented by corresponding variations in the value of the parameter α_0, *i.e.* in the "basic" abnormality or susceptibility of the individual corresponding to a zero dosage [*i.e.* putting $x_0 = 0$ in equation (1)]. As in many other fields of application in which similar techniques are applied (Finney, 1952), it is assumed that the individual values of this parameter follow a Gaussian (normal) distribution, with mean α and variance σ^2. Thus, α can be regarded as the average value of α_0 in the population and σ^2 is a measure of the variability of the abnormality–exposure relationship from individual to individual. It should be noted that β is assumed to be constant throughout the whole population. This implies that the variability of the abnormality values (y_0) is independent of the applied dosage.

The Effect of Errors of Measurement.—The above analysis is based on the assumption that the true values of abnormality and dosage are known exactly. In fact this is not so, and it is necessary to take account of the errors of measurement.

The measurement of abnormality depends on the subjective assessment of a chest radiograph, with all the attendant uncertainties both in technique and interpretation. Fay and Ashford (1960) have analysed the errors associated with the classification of radiographs for simple pneumoconiosis. Their treatment is based on the concept of an "*x*-ray reading" scale of radiological abnormality, which is chosen in such a way that the readings (y') on any given film follow a Gaussian distribution about the true abnormality value (y) with characteristic variance ρ^2, which is independent of y. This scale is defined so that the boundaries between categories 0 and 1 and categories 1 and 2 occur respectively at the points zero and unity. Although the *x*-ray reading scale has these two points in common with the "abnormality" scale defined above, there is no *a priori* reason why the two scales should be the same. Analyses of the data obtained on the medical surveys suggest, however, that the *x*-ray reading and abnormality scales may be regarded as coincident, at least in the vicinity of the "0-1" and "1-2" boundaries. Under this assumption Ashford (1959) has shown that the effect of unbiased reading errors

is to increase the variance of the observed relationship between abnormality and exposure to $(\sigma^2 + \rho^2)$, leaving the α and β parameters unchanged. Very little is known about the errors associated with taking the radiograph, but what evidence is available within the Pneumoconiosis Field Research suggests that they may be incorporated with the reading errors, and thus tend to inflate the value of ρ^2 without leading to any appreciable change in the form of the error distribution.

The measurement of the dosage is also subject to error and the estimates applied in the analyses are assumed to follow a Gaussian distribution about the true values, with variance τ^2. The effect of these errors (Ashford, 1959) is further to increase the variance of the observed relationship to $(\sigma^2 + \rho^2 + \beta^2\tau^2)$, again leaving the α and β parameters unchanged.

Thus, provided the errors of measurement are made without bias and follow a Gaussian distribution, their effect is merely to increase the apparent variability of the individual responses, without changing the basic abnormality–response relationship.

The Quantal Relationship.—The approach developed in the previous paragraphs amounts to the assumption that there exists a basic relation between the observed values of the abnormality (y') and the dosage (x') such that y' follows a Gaussian distribution with mean $(\alpha + \beta x')$ and effective variance σ'^2 $(= \sigma^2 + \rho^2 + \beta^2\tau^2)$. This means that for a given observed value of the dosage (x') the observed abnormality values will follow a characteristic bell-shaped Gaussian distribution, as illustrated in Fig. 2.

As we are dealing with a statistical distribution (*i.e.* as the man's abnormality must have some value, large or small), the total area under the Gaussian curve must be unity. If a man selected at random from the population is exposed to an observed dosage (x') the probability that his observed abnormality (y') will exceed any particular point y_i on the scale is represented by the shaded area under the curve to the right of y_i. In mathematical terms,

$$\text{Prob.}\,(y' \geqslant y_i) = \int_{\frac{y_i - \alpha + \beta x'}{\sigma'}}^{\infty} \phi(t)\,dt = \Phi\left\{\frac{\alpha + \beta x' - y_i}{\sigma'}\right\} \quad . \qquad (2)$$

where $\phi(t)$ and $\Phi(t)$ denote respectively the frequency function and the cumulative probability function corresponding to the standardized Gaussian distribution. Reference to Fig. 1 shows that the probability that the abnormality value will exceed the boundary between categories 0 and 1 (*i.e.* that the film will be read as category 1 or more) may be written,

$$\text{Prob.}\,(y' \geqslant 0) = \Phi\left\{\frac{\alpha + \beta x'}{\sigma'}\right\} \quad . \qquad (3)$$

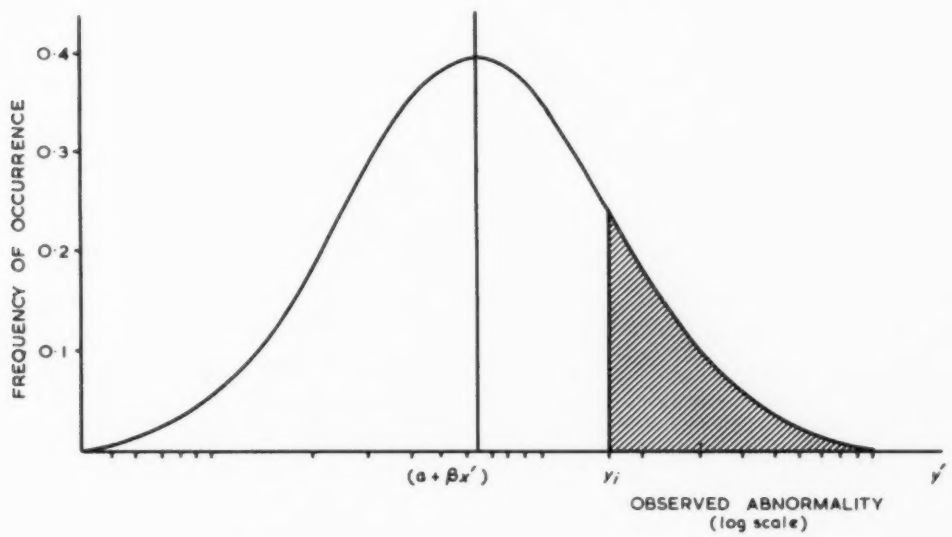

FIG. 2.—Distribution of abnormality for a given observed dosage.

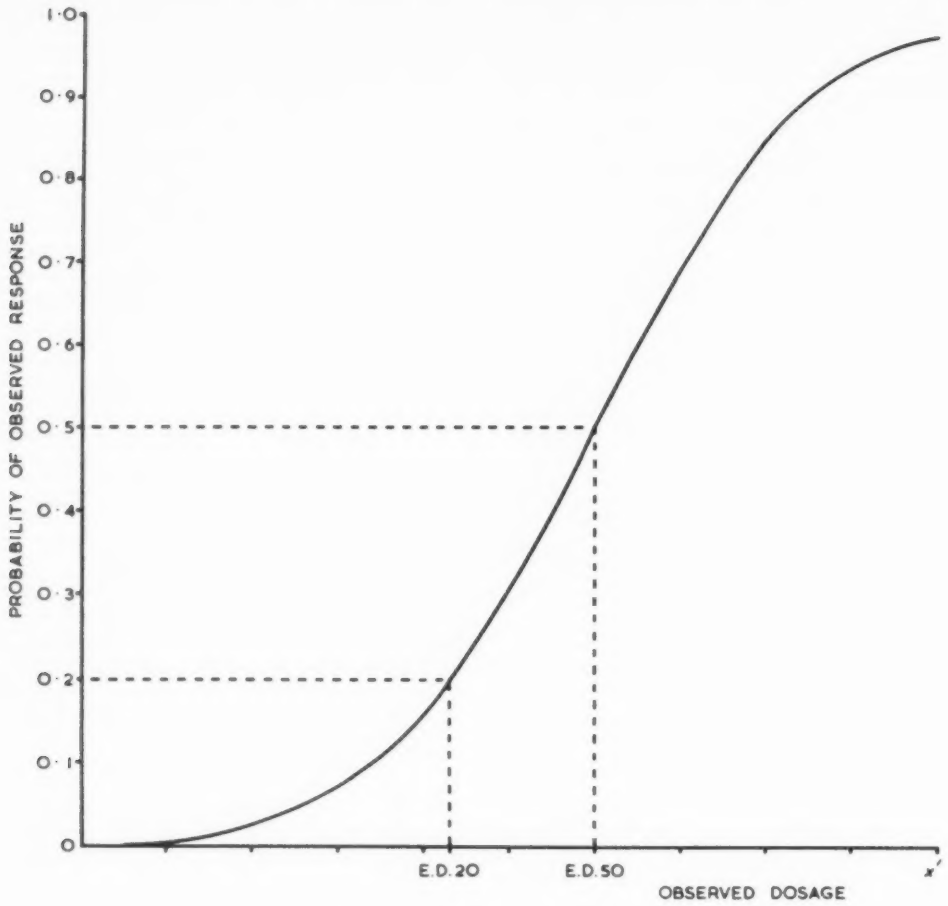

FIG. 3.—A typical sigmoid relationship.

Similarly the probability that the abnormality value will exceed the boundary between categories 1 and 2 (*i.e.* that the film will be read as category 2 or more) may be written,

$$\text{Prob.}\ (y' \geq 1) = \Phi\left\{\frac{\alpha + \beta x' - 1}{\sigma'}\right\} \quad . \qquad (4)$$

In practice the abnormality values can only be observed in terms of the discrete categories, *i.e.* in terms of the proportion of men with a given dosage who have developed a given category of pneumoconiosis or more. This means that the data on which studies of abnormality and dosage are based must take the form of the quantal relations (3) and (4), instead of the direct quantitative relation (1). Both the quantal relations involve the same basic parameters α, β, and σ', and Ashford (1959) has shown how these may be estimated from the quantal data, and hence how the underlying quantitative relation may be deduced. Briefly, the method depends upon expressing the probabilities that a man's film is read as category 0, category 1, or category 2 or more, in terms of expressions (3) and (4). On this basis the likelihood of any given set of observations may be expressed as a function of the parameters α, β, and σ', and the best estimates of the values of these parameters derived by the method of maximum likelihood.

Equation (3) relates the probability of the man's radiograph being classified as category 1 or more (*i.e.* of response) to the observed dosage. When the probability is plotted against dosage the relation takes the characteristic form of an S-shaped curve as shown in Fig. 3. This type of dosage–response curve is familiar in many branches of biological research, and Roach (1953) has referred to the

possible application of the general method to the study of coalworkers' pneumoconiosis.

The probability of response increases smoothly from zero for small values of the dosage to unity for large values. The effective dosage (E.D.) values corresponding to a 20% and 50% probability of response are denoted respectively the E.D. 20 and E.D. 50, and these values are frequently used to provide an assessment of the relation.

For the purposes of the statistical analysis it is more convenient to work in terms of a linear relationship, and it is the accepted practice (Fechner, 1861; Bliss, 1935) to adjust the probability scale to achieve this effect, by means of the "probit" transformation. When this is done the probit* (p) of the probability of response (P) is related to the observed dosage as shown in Fig. 4. This relation between probit and dosage provides a convenient summary of the effect of exposure on abnormality and thus provides a measure of the hazard. As it is linear in form only two parameters (position and slope) are involved, and these reflect the three parameters α, β, and σ' associated with the underlying abnormality–exposure relationship. Although the dosage–response relation shown in Fig. 4 is derived from the underlying abnormality–exposure relation given in Fig. 1, it must be emphasized that the former is concerned only with the chance of exceeding some given point on the abnormality scale, whereas the latter deals with the abnormality value itself.

For the category 1 or more response the linear relation takes the form,

$$p = \frac{\alpha + \beta x'}{\sigma'} \quad . \qquad (5)$$

The point at which this line crosses the x'-axis $[\alpha/\beta]$ corresponds to a 50% probability of response, and is denoted the E.D. 50. The position of this point depends only on the position and slope of the underlying abnormality–exposure relation and is independent of the variability of the individual susceptibilities and of the errors of measurement. The slope of the quantal response line is β/σ' and is thus directly proportional to the slope of the underlying response line and inversely proportional to the effective variability of the individual susceptibilities. Any reduction in the errors of measurement or in the individual variation within the population would lead to a corresponding increase in the slope of the quantal response line. In the limit the

*This is defined by the relation:—$P = \Phi(p)$.

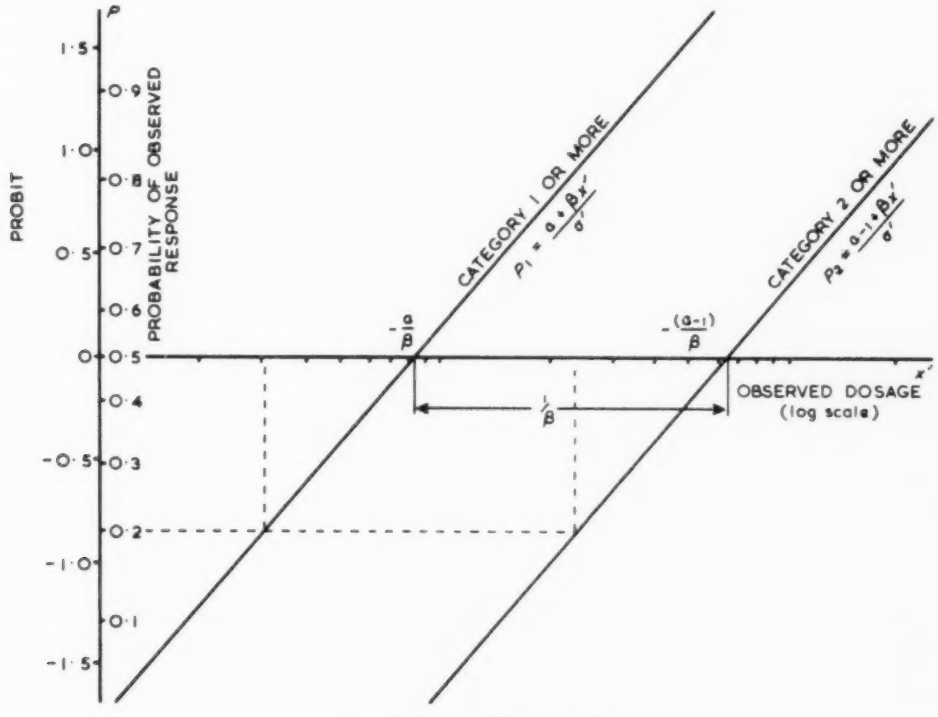

FIG. 4.—A typical probit relationship.

quantal response line tends to become parallel to the p-axis as the effective variability tends towards zero. Under these conditions there would be a constant "threshold" dose for the whole population and the sigmoid curve illustrated in Fig. 3 would become a "step" function.

If we consider two different response levels say $P_a\%$ and $P_b\%$, the difference between the dosages corresponding to the E.D. P_a and the E.D. P_b will be, from (5),

$$x_a - x_b = \frac{\sigma'(p_a - p_b)}{\beta} \qquad (6)$$

and will thus depend on β and σ', and not on α. As the dosage is measured on a logarithmic scale this means that the ratio of the E.D. P_1 to the E.D. P_2 will depend on the effective variance σ', as well as on P_1 and P_2.

Reference to equations (3), (4), and (5) shows that the relation between the probit of the probability that the subject develops category 2 or more pneumoconiosis and dosage takes the form,

$$p = \frac{\alpha - 1 + \beta x'}{\sigma'} \qquad . \qquad (7)$$

Thus the quantal response lines for category 1 or more and category 2 or more pneumoconiosis are parallel, the former lying to the left of the latter (Fig. 4). The horizontal difference between the two lines (*i.e.* the difference in dosage values corresponding to any given probability of response) is equal to the reciprocal of the slope of the underlying abnormality–dosage relationship, $1/\beta$. As dosage is measured on a logarithmic scale the ratio of corresponding E.D's for category 1 or more and category 2 or more pneumoconiosis is therefore a constant for all response levels and all analyses dealing with the same population, regardless of the errors.

Measures of Exposure.—At the present time there are many uncertainties about which particular aspects of environmental exposure are of importance in the development of simple pneumoconiosis (Fay and Hicks, 1960). Indeed it will be one of the main tasks of the Pneumoconiosis Field Research to clarify the situation. In the absence of definite information about the nature of the hazard there are several possible measures of exposure which might be considered, each reflecting a different hypothesis about the relation between abnormality and exposure. Examples are (i) total particle number exposure in the 1-5 micron size range [expressed as the integrated product of average number concentration (all dust, 1-5 microns) and time at risk], (ii) total surface area exposure in the 1-5 micron size range, (iii) quartz exposure in terms of the surface area of particles in the 0·5-5 micron size range, and so on. Measures of exposure of this kind involve a detailed knowledge of the composition and concentration of the airborne dust particles throughout the whole of the working life of the man examined. This type of information is not available for the period before the first medical surveys at the selected collieries. It is therefore necessary to turn to less refined measures based on the men's working histories, in terms of the periods spent in particular occupations or types of environment, to represent the man's exposure up to the time of the first medical survey.

To illustrate the use to which the industrial history data may be put we will consider the prevalence of pneumoconiosis amongst a group of men who have worked for varying periods in one specific occupation. If the prevailing level of the particular type of hazard which is relevant to the causation of pneumoconiosis (denoted I) has remained constant throughout the whole period, the total exposure of a man who has followed the occupation for a time t may be represented by the product $I \times t$. His "dosage" (*i.e.* the logarithm of his total exposure) will then be

$$x = \log(I \times t) = \log I + \log t \qquad . \qquad (8)$$

Reference to equation (1) shows that the underlying dosage–abnormality relation takes the form,

$$y = (\alpha + \beta \log I) + \beta \log t \qquad . \qquad (9)$$

It follows that there is an equivalent abnormality–time relationship, and that log time may be regarded as a substitute for the true dosage, which is unknown. Comparison of equations (1) and (9) shows that the slope of the corresponding response lines remains unchanged, although the constant term α, which represents average susceptibility, is increased by a factor $\beta \log I$ (*i.e.* by a factor proportional to the logarithm of the mean exposure level).

Even if adequate quantitative and qualitative data are available about environmental conditions, the precise combination of these measurements which represents the true hazard is unknown. Analyses in terms of measured exposure must therefore be directed towards the testing of certain hypotheses about the nature of the hazard and it is unlikely that, on any one occasion, the hypothesis chosen will correspond exactly to the true situation. This means that, if a particular hypothesis H is under investigation, the measurements of dosage x_H based on this hypothesis will not correspond exactly to the true measurements of dosage x expressed in the correct terms. For this reason the dosage values x_H used in the analysis must be regarded as estimates of the true dosages x. On the assumption that the x_H values follow a Gaussian

distribution with mean $(x + \lambda_H)$ and variance τ_H^2, the underlying relation between abnormality and exposure (when expressed in terms of hypothesis H) will take the same form as the corresponding relation between abnormality and period of exposure given at equation (9), the only difference being that λ_H is substituted for $\log l$ and the dosage x_H is substituted for the logarithm of the time spent in the particular occupation. The abnormality–exposure relation when time is taken as a measure of exposure (amongst a group of men who have been exposed to only a single level of the hazard) is therefore equivalent to the relation when exposure is measured in terms of some particular hypothesis H, unless this hypothesis happens to provide an exact measure of the hazard (when both λ_H and τ_H^2 will vanish).

For the type of analysis under consideration the values of the dosage x_H are obtained by sampling methods and are not therefore known exactly. The estimated values x'_H are found to follow a Gaussian distribution about x_H with variance $\tau_H'^2$. Thus, the effective variance of the x'_H about the true dosage x will be $(\tau_H^2 + \tau_H'^2)$. If two hypotheses are compared, the better (*i.e.* the one which gives the closer approximation to the true hazard) will be the hypothesis associated with the smaller value of τ_H^2.

Comparison of Hazard Associated with Different Occupations.—If a population of men is assigned at random to one or other of two occupations in which they spend the remainder of their working lives up to the time of the medical examination, and if the mean levels of the hazard associated with the occupations are denoted l_a and l_b, the corresponding abnormality–time relationships may be written,

$$y_a = (\alpha + \beta \log l_a) + \beta \log t_a \qquad (10)$$

$$\text{and } y_b = (\alpha + \beta \log l_b) + \beta \log t_b \quad . \qquad (11)$$

The variances of the individual abnormality values are respectively,

$$\sigma_a'^2 = \sigma^2 + \rho^2 + \beta^2 \tau_a^2 \qquad (12)$$

$$\text{and } \sigma_b'^2 = \sigma^2 + \rho^2 + \beta^2 \tau_b^2 \quad , \qquad (13)$$

where τ_a^2 and τ_b^2 are associated respectively with the variances of the errors in the $\log t_a$ and $\log t_b$ measurements. In general, τ_a^2 and τ_b^2 (and hence σ'^2_a and σ'^2_b) will be different.

Equation (5) shows that the corresponding quantal relationships for category 1 or more pneumoconiosis are,

$$p_a = \frac{1}{\sigma'_a}[(\alpha + \beta \log l_a) + \beta \log t_a] \qquad (14)$$

$$\text{and } p_b = \frac{1}{\sigma'_b}[(\alpha + \beta \log l_b) + \beta \log t_b] \, . \qquad (15)$$

The two lines will, in general, differ both in position (unless $l_a = l_b$ and $\tau_a = \tau_b$) and slope (unless $\tau_a = \tau_b$). Because the slopes are not necessarily the same the lines may cross, and comparisons between the relative hazards associated with the two occupations in terms of the quantal response lines themselves may be ambiguous. In other words, the usefulness of the quantal response lines for the comparison of the hazards associated with two different occupations is limited by the differential effect of errors in the dosage variable. On the other hand, the analysis in terms of the underlying quantitative response, to obtain estimates of $\beta, \sigma'_a, \sigma'_b, (\alpha + \beta \log l_a)$, and $(\alpha + \beta \log l_b)$ [equations (10)-(13)] will provide direct information about the ratio (l_a/l_b) of the levels of hazard associated with the two occupations.

A Typical Analysis.—An example of the application of the methods described above is provided by an analysis of the data obtained on the first medical survey at a particular colliery for a group of men who had previously worked only in a single specified occupation. To reduce the calculations to manageable proportions the 371 men concerned were divided into eight groups according to period spent in the occupation, and the numbers of men in each group whose radiographs were classified as category 0, category 1, and category 2 or more were recorded. In theory each individual could have been regarded as defining a separate group, but a considerable saving can be made in the calculations without any appreciable effect on the final results if the men with similar exposures are grouped together for the analysis. On the basis of the numbers of men with category 0, category 1, and category 2 or more pneumoconiosis in the various groups, the quantal relations between period spent in the occupation and the chance of developing category 1 or more and category 2 or more pneumoconiosis were calculated, and the underlying abnormality–exposure relation was derived.

In order to test whether the observed data are consistent with the assumptions on which the calculations are based, the expected numbers of men assigned to the various radiological categories (predicted on the basis of the estimated abnormality–exposure relation) were compared with the corresponding observed results. Details are given in the Table which shows that agreement between theory and observations is good. The χ^2 value, after grouping the first three and last two entries in the Table, is 4·62, which for 7 degrees of freedom is not significant ($p \approx 0{\cdot}7$).

The corresponding quantal and quantitative relations are illustrated respectively at Figs. 5a and b. It should be noted (Fig. 5a) that the quantal response lines corresponding to category 1 or more and

TABLE

PERIOD OF EXPOSURE AND PREVALENCE OF PNEUMOCONIOSIS AMONGST A GROUP OF COAL-MINERS WHO HAVE WORKED PREDOMINANTLY IN THE SAME OCCUPATION

Mean Period Spent (years)	Number of Men					
	Category 0		Category 1		Category 2 or More	
	Observed	Expected	Observed	Expected	Observed	Expected
5·8	98	97·5	0	0·4	0	0·1
15·0	51 183	49·2 181·6	2 8	3·6 9·4	1 4	1·3 4·1
21·5	34	34·9	6	5·4	3	2·7
27·5	35	34·3	5	8·3	8	5·5
33·5	32	31·7	10	10·6	9	8·7
39·5	23	20·5	7	8·8	8	8·7
46·0	12	12·9	6	6·9	10	8·2
51·5	4 16	4·5 17·4	2 8	2·8 9·7	5 15	3·8 12·0

category 2 or more pneumoconiosis are parallel, as required by the theoretical treatment. For this reason it is possible to obtain more reliable estimates of the quantal relations than could be derived if the corresponding relations for category 1 or more and category 2 or more pneumoconiosis were calculated separately. A further advantage as compared with the conventional quantal response analysis is that the underlying relation between abnormality and exposure provides a better description of the natural history of simple pneumoconiosis and enables conclusions to be drawn about the chance of developing any specified degree of pneumoconiosis, whether or not it forms the boundary of two of the standard

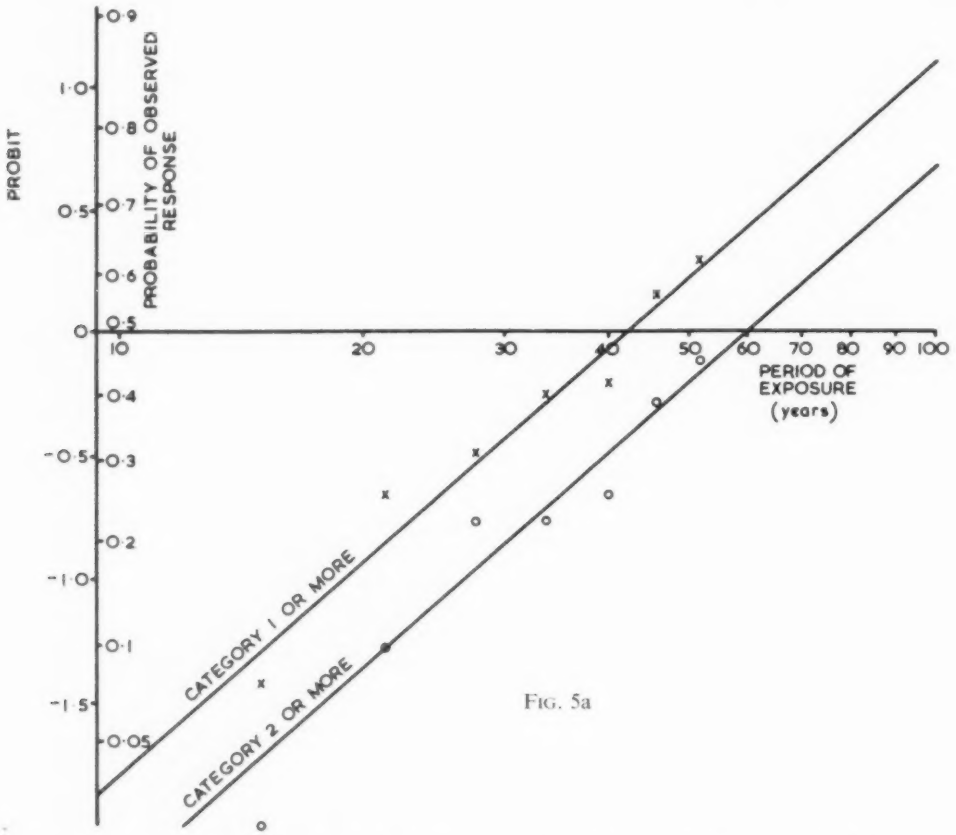

FIG. 5a

FIG. 5.—Analysis of the prevalence of pneumoconiosis amongst a group of men who have worked only in one particular occupation. (a) The quantal response; (b) (Opposite) The quantitative response.

categories. For example, for a period of exposure of 20 years it is found that the corresponding observed abnormality value y' will follow a Gaussian distribution, with mean $-1{\cdot}52$ (Fig. 5b) and standard deviation 1·56. Thus by reference to tables of the Gaussian distribution it can be deduced that the chance of the abnormality value exceeding zero (*i.e.* of the man being classified as category 1 or more) is approximately one-sixth. The new approach also provides an estimate of the combined variability of the individual susceptibilities and the errors of measurement; in this particular analysis we have seen that the calculated value of the standard deviation of the combined error is 1·56.

The Effect of "Mixed Exposures"

Method of Approach.—The approach described above provides a satisfactory basis for estimating the relation between abnormality at the time of the first medical survey and previous environmental exposure, subject to the *proviso* that this latter measurement can be made in terms of a single variable (*i.e.* that the man has worked only in one occupation or class of environment). Unfortunately, it is usual in the mining industry for men to follow a variety of different jobs during the course of their working lives, thereby being exposed to a number of different levels of hazard. It is the normal practice for a new recruit to start work on the surface, or at non-facework underground, and to progress after a period (depending on local custom and circumstances) to the more highly paid occupations on the coal-face. Generally speaking, a coal-face worker will continue to work on the coal-face until the decline of his physical powers as a result of increasing age or of other causes makes it necessary for him to transfer to less arduous (and less remunerative) employment away from the coal-face, either underground or on the surface.

When the analysis of the results of the first round of medical surveys was carried out it would have been most unfortunate to have had to exclude the large proportion of men with "mixed" industrial histories. Consideration was therefore given to the development of a method of analysis to cope with the situation in which the man's past environmental exposure must be measured in terms of the periods spent in two or more specific occupations or classes of environment. Following the method of approach described above for dealing with exposure at a single

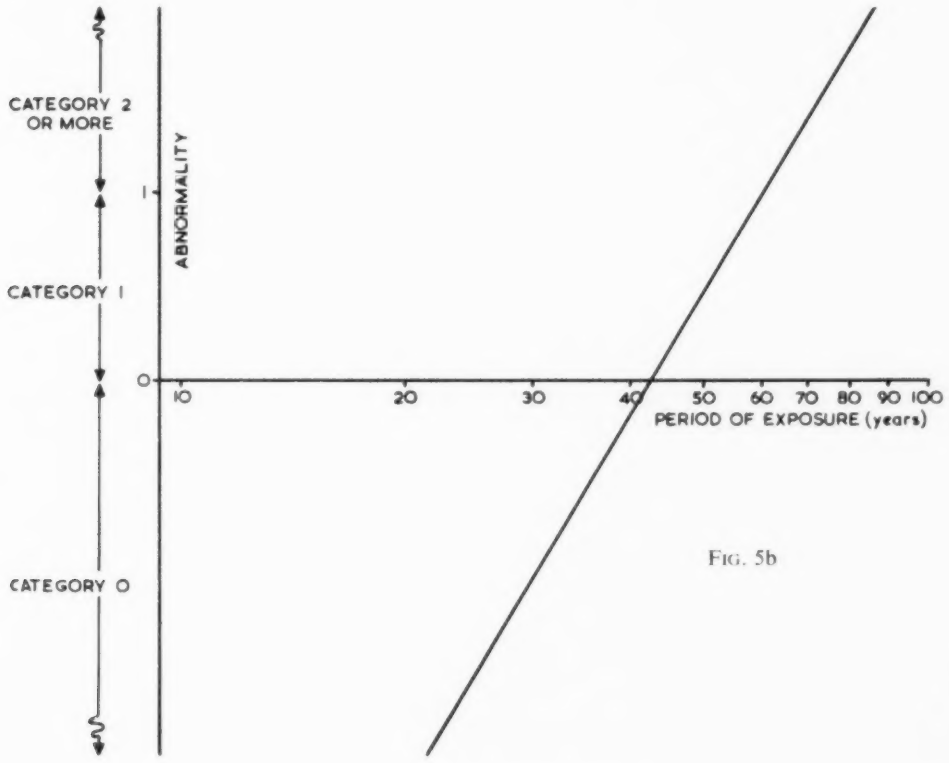

FIG. 5b

level of the hazard, we will assume that a man has spent periods t_1 and t_2 in two distinct occupations or classes of environment E_1 and E_2. The prevailing levels of the hazard (assumed to be constant) are denoted l_1 and l_2 ($= kl_1$). Thus, environment E_2 is assumed to be k times as hazardous as environment E_1. The man's total exposure will consist of the sum of his exposures in each of the two environments separately. In these circumstances his total dosage (*i.e.* the logarithm of his total exposure) will be given by the expression,

$$x = \log (t_1 l_1 + t_2 l_2)$$
$$= \log l_1 + \log (t_1 + kt_2) \quad . \quad (16)$$

The abnormality–exposure relation will take the form,

$$y = (\alpha + \beta \log l_1) + \beta \log (t_1 + kt_2) \quad . \quad (17)$$

Comparison with equation (9), which describes the corresponding relation for exposure in a single environment, shows that the effective dosage can be taken as $\log (t_1 + kt_2)$. It follows that the variance of the Gaussian distribution of the observed individual responses is given approximately by the expression,

$$\sigma'^2 = \sigma^2 + \rho^2 + \beta^2 \left[\frac{t_1^2 \tau_1^2 + k^2 t_2^2 \tau_2^2}{(t_1 + kt_2)^2} \right], \quad (18)$$

where τ_1^2 and τ_2^2 are the variances associated with the measurements of the individual dosages $\log t_1$ and $\log t_2$.

By means of equations (17) and (18) the quantal response relationships based on the underlying quantitative response can be expressed in terms of a "multi-dimensional" exposure (t_1, t_2) in the same way as is done for a single-dimensional exposure. Ashford (1958a) has shown how an approach of this type may be applied to obtain information, from the "mixed" exposure data, about the dosage–abnormality relation associated with each of the constituent hazards separately. In this connexion it should be noted that the presence of errors in the estimates of the time spent in the various classes of environment (which leads to the expression (18) for the effective variance of the individual responses) means that the quantal relationships between the probability of response and the multi-dimensional exposure cannot be derived directly from the quantal relations between the probability of response and each of the dimensions of exposure separately.

Applications.—Analysis of the results of the first round of medical surveys at the selected collieries (Hicks *et al.*, 1961) has shown that there are, in general, four main types of environment which involve an appreciable pneumoconiosis hazard, namely hard heading (*i.e.* tunnelling in rock), coal-face (coal-getting shift), coal-face (preparation shift), and elsewhere underground. The hazard associated with work on the surface can, in general, be regarded as negligible. It is also found that only a very small proportion of the men examined have had experience of hard heading and, if these men are excluded from the analysis, each man's exposure can be regarded as a three-dimensional variable corresponding to the periods spent in each of the three remaining environments.

Thus, if a man has spent t_1 years on the coal-face (coal-getting shift), t_2 years on the coal-face (preparation shift) and t_3 years elsewhere underground, his total exposure may be summarized in terms of the three-dimensional variable (t_1, t_2, t_3). In this particular application of the multi-dimensional method (although this is not necessarily so in general) all periods of exposure in a given environment are taken together, whether or not they refer to the same working places or even to the same colliery. This approach involves the tacit assumption that each separate environment is associated with the same level of hazard for all working places. This assumption can be justified as a reasonable working approximation for a number of reasons. In the first place it is found that the majority of men have worked only at the colliery at which they were examined or at other collieries in the immediate vicinity. Secondly, studies carried out by the Pneumoconiosis Field Research have shown that conditions do not, in general, vary greatly for the same environment (a) from colliery to colliery in the same locality, (b) from one working place to another within the same colliery, or (c) from time to time at the same working place, provided that the methods of working are similar. Thirdly, the greater part of the men's exposure before the first medical surveys, took place during a period when methods of working were relatively stable, before the introduction of power loading on an appreciable scale. The final test of the validity of the assumption may be obtained by comparing the observed numbers of men with the various categories of pneumoconiosis with the numbers predicted on the basis of the assumed model; for a large number of analyses the agreement has been found to be extremely good (Hicks *et al.*, 1961), and it can be asserted with confidence that the observed data are consistent with the theoretical treatment.

At one particular colliery the periods spent by each man (excluding those who had ever worked as hard headers) on the coal-face (coal-getting shift), coal-face (preparation shift), and elsewhere underground were calculated from his past working history. In order to reduce the magnitude of the mathematical calculations, the population (of some 1,700 men) was subdivided into about 50 different

groups, each group including men with similar periods of exposure in each of the three main classes of environment. On the basis of the numbers of men in each group with category 0, category 1, and category 2 or more and the corresponding average periods of exposure in the three environments, the quantal response lines for category 1 or more pneumoconiosis and category 2 or more pneumoconiosis and the corresponding underlying abnormality–exposure relations were calculated for each class of environment separately. The mathematical calculations involved have been described in detail by Ashford (1958a; 1959). The resulting quantal response lines are shown at Fig. 6a and the underlying quantitative relationship appears at Fig. 6b.

Fig. 6a shows that, as required by the model, the quantal response lines from category 1 or more and category 2 or more pneumoconiosis for each of the environments separately are parallel. When the individual category 1 or more lines are considered, however, comparisons of the hazard associated with the three environments are ambiguous in the sense that, for example, work elsewhere underground appears to have been more hazardous for short periods of exposure and less hazardous for long periods of exposure than work on the coal-face. This ambiguity is a reflection of the fact (discussed above) that the errors associated with the use of the

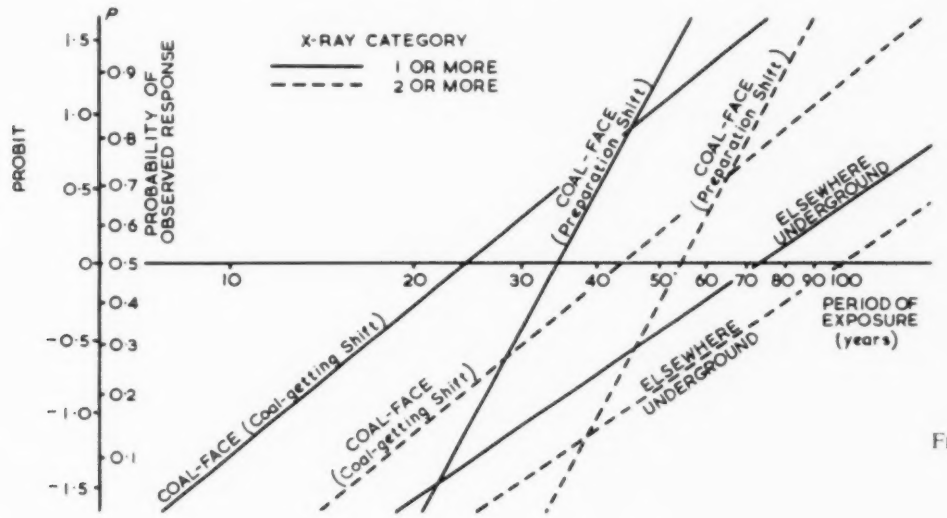

FIG. 6a

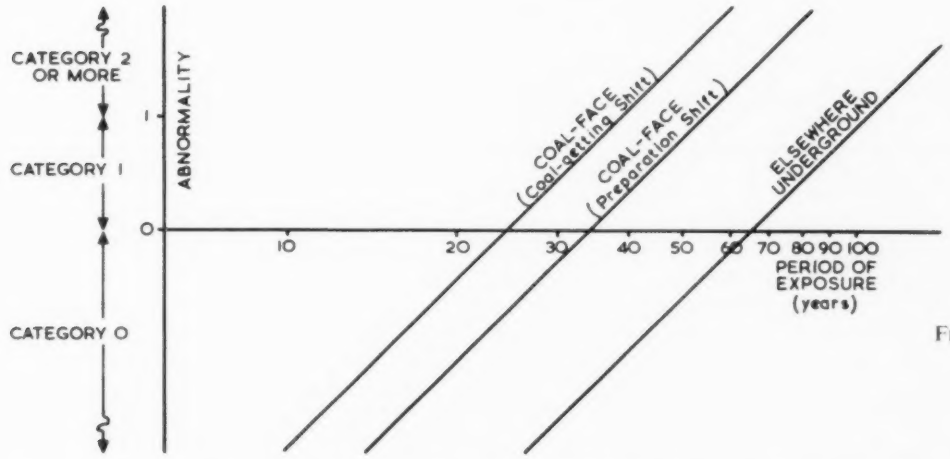

FIG. 6b

FIG. 6.—Multi-dimensional analysis of the prevalence of pneumoconiosis in a colliery population in terms of the periods spent on the coal-face (coal-getting shift), coal-face (preparation shift), and elsewhere underground. (a) The quantal response; (b) The quantitative response.

period of exposure in any given environment as a measure of the hazard differ from environment to environment and that, in consequence, the observed quantal response lines are not, in general, parallel.

Consideration of the underlying abnormality–exposure relations enables the effect of the errors to be treated separately (Fig. 6). It will be seen that the abnormality–exposure lines corresponding to the three environments separately are parallel, as required by the method of analysis, and that the ambiguity associated with comparisons of the relative hazards in terms of the quantal response lines is now removed. As exposure is measured on a logarithmic scale, the ratio of the periods spent in any pair of the three environments leading to any particular abnormality value is constant and corresponds to the relative hazard. In this analysis the hazard on the coal-face (coal-getting shift) was about four-thirds of that on the coal-face (preparation shift) and about eight-thirds of that for exposure elsewhere underground. The standard deviations of the observed susceptibilities (which represent the summation of the variability associated with the true distribution of susceptibilities, the errors of taking and reading the radiographs, and the errors associated with the use of time as a measure of exposure) were respectively 1·5, 0·72, and 1·8 for the coal-face (coal-getting shift), coal-face (preparation shift), and elsewhere underground. These differences are a reflection of differences between the three environments in the errors associated with the use of time as a measure of exposure.

To summarize, the multi-dimensional approach has provided the means of estimating the past hazard associated with each of the three main environments separately, on the basis of the mixed working history data from all the men examined at a colliery, excluding only the small number of men who have worked as hard headers. This is only one of a number of applications of the method, which has also been used (a) to compare the hazards associated with two different occupations on the basis of groups of men who have worked mainly in one or other or both of the two occupations, (b) to compare the hazard associated with the colliery at which the men were examined with the average hazard at the other collieries at which they have worked in the past, on the basis of the corresponding periods spent in all dusty occupations, and (c) to analyse the data from successive medical surveys, as described below.

Analysis of the Progression Data

The Measurement of Progression.—Each successive round of medical surveys will provide information about the man's radiological appearance at the corresponding point in time. The classification of simple pneumoconiosis is made in terms of a small number of discrete categories, and inevitably each category covers a considerable range of abnormality. Hence, it is not possible to obtain any useful measure of progression merely by subtracting the "earlier" from the "later" reading, as would be the case if the measurements of abnormality could be made with precision on a continuous and uniform scale. There is the further complication that the radiograph readings are subject to error, which means that a man may be assigned to a higher category on the earlier survey than on the later one, if the readings are made independently.

The possibility of making use of the "average" of a number of independent readings to obtain a more reliable subdivision of the abnormality scale has been under active consideration since the start of the Research. There are, however, several inherent difficulties. In the first place the number of different sets of readings which can be obtained on each film is limited by the effort available; this point is particularly important when upwards of 30,000 men are involved. On the first round of medical surveys three such sets of readings were obtained at most. By averaging these three readings, each category can be divided into three (not necessarily equal) parts, but these sub-categories themselves cover a considerable range of abnormality. With the comparatively small amount of progression which has so far been found between the first and second medical surveys this subdivision would by no means provide an adequate substitute for a continuous scale. Secondly, a detailed study of the effect of "averaging" (Fay and Ashford, 1960) has shown that the position of the sub-categories so obtained depends on the errors associated with the individual sets of readings, and Ashford (1960) has shown that the magnitude of these errors may vary widely from one set of readings to another. In theory, a suitable correction for the individual reading errors could be applied, but the necessary calculations would be so complex as to cancel out the apparent simplicity of the method.

An alternative approach was therefore considered. This involved the examination of all the films obtained for each man (on successive surveys) at the same time; if any change in the characteristic abnormalities associated with simple pneumoconiosis was observed (after due allowance had been made for radiographic technique) the man was to be classified as showing evidence of "progression" (or "regression"). The fundamental disadvantages of this procedure are, first, that no quantitative information can be obtained of the change in abnor-

mality associated with a "progression" (or "regression") reading and, secondly, that there is no certainty that any given amount of radiological change will be equally evident at all points on the abnormality scale.

For these reasons it was decided to consider the problem in terms of the changes (if any) in radiological category between the man's first exposure to airborne dust (when it is reasonable to assume that his radiological abnormality value will be at the lower limits of category 0) and the times of the successive medical surveys. This approach demands a knowledge of the man's radiological category at each medical survey, so that the position of his corresponding radiological abnormality value relative to the various category boundaries can be determined. The definitive readings have been shown (Ashford, 1960) to be free from systematic bias and therefore provide a satisfactory measure of radiological category for this purpose. There are two possible general procedures for obtaining the definitive radiological readings on a series of films taken at different points in time. On the one hand the films for successive surveys may be read independently, or, alternatively, the corresponding films on successive surveys may be examined side-by-side, the reading of each film being subject to influence by the appearance of the others. At the present time the relative merits of the two methods are being assessed within the Research, by means of a series of film reading trials.

The Measurement of Exposure.—If a man starts at the lower limit of category 0 on entering the mining industry, his radiological appearance at any given time will depend on his subsequent exposure. We have seen that each man's exposure at the time of the first medical survey may be expressed as a multi-dimensional variable corresponding to the periods he has spent in each of the main classes of environment. At the time of the second medical survey the man has been subject to a further ("measured") exposure, and his cumulative exposure since joining the mining industry can also be regarded as a multi-dimensional variable in terms of the periods spent in the main classes of environment up to the time of the first survey, with one or more additional dimensions corresponding to his "measured" exposure between the first and second surveys. For example, the man's total exposure up to the time of the second medical survey might possibly be represented by a four-dimensional variable, in terms of the periods spent before the first survey on the coal-face (coal-getting shift), coal-face (preparation shift), and elsewhere underground, and of his "measured" exposure between the two surveys. It has been shown that the use of the period of exposure in a given occupation as a substitute for true exposure is based on the assumption that the dosage corresponding to a period t spent in an occupation with mean hazard l is given by the expression,

$$x = \log (l \times t) = \log l + \log t \quad .$$

If the mean levels of hazard associated with the three types of environment are denoted l_1, l_2, and l_3, and if the periods spent in these environments up to the time of the first medical survey are denoted t_1, t_2, and t_3 respectively, the man's dosage before the first medical survey is therefore,

$$x = \log [(l_1 \times t_1) + (l_2 \times t_2) + (l_3 \times t_3)] \, . \quad (19)$$

His "measured" component of exposure, denoted y, between the first and second surveys will take the form of the sum of a series of products (corresponding to each of the occupational groups to which the man has belonged) of the mean hazard (expressed in accordance with the particular hypothesis about exposure which is under examination) and the corresponding period of exposure. If the levels of hazard l_1, l_2, and l_3 are expressed in the same form as the levels used for the calculation of the "measured" exposure, all four components of the man's exposure up to the time of the second survey will be expressed in the same terms. The man's total dosage then takes the form,

$$x = \log [(l_1 \times t_1) + (l_2 \times t_2) + (l_3 \times t_3) + y] \, . \quad (20)$$

At first sight, the use of a multi-dimensional variable expressed in terms of a mixture of time and "measured" exposure might appear to be subject to criticism, but reference to expression (20) shows that in fact like is being added to like, and it therefore follows that the procedure is valid.

It can also be shown that the variance of the Gaussian distribution of individual exposures is given approximately by the expression,

$$\sigma'^2 = \sigma^2 + \rho^2 + \beta^2 \left[\frac{l_1^2 t_1^2 \tau_1^2 + l_2^2 t_2^2 \tau_2^2 + l_3^2 t_3^2 \tau_3^2 + y^2 \tau^2}{(l_1 t_1 + l_2 t_2 + l_3 t_3 + y)^2} \right], \quad (21)$$

where τ_1^2, τ_2^2, τ_3^2, and τ^2 are respectively the variances associated with the measurements of $\log t_1$, $\log t_2$, $\log t_3$, and y respectively. It should be noted that the variance τ^2 of the "measured" component of dosage represents a summation of the errors of estimation associated with the particular measure of hazard chosen and of the errors associated with the choice of this particular measure of hazard as a representation of the man's true exposure.

By means of the underlying abnormality–exposure relation (1) and expressions (19), (20), and (21) the

probability that a man has developed any given category of simple pneumoconiosis or more at the time of each successive medical survey may be expressed in terms of the two sets of multi-dimensional variables. Hence, the underlying abnormality–exposure relations for each of the dimensions separately may be estimated, and the effect of the "measured" component of exposure may be assessed. The relative accuracy of the resulting estimates will depend on the proportion of the man's total exposure which is accounted for by the "measured" component. The information about the relation between abnormality and the "measured" component of exposure will therefore become progressively more precise as the number of surveys increases.

An Example of the Method.—The proposed method may be illustrated by the analysis of the data obtained at one particular colliery between the first and second medical surveys. On this occasion the radiographs obtained on the surveys were classified independently and, by reading a check sample of first survey films again on the second survey, it was established that there had been no significant change in the levels of classification of the definitive readings.

The population considered for the progression analysis consists of all men examined on both first and second medical surveys with no record of exposure to airborne dust other than at the colliery during the intervening period. The following data are available for each man:

(i) Independent definitive readings of his chest radiographs on the two successive surveys, in terms of the I.L.O. (1953) classification;

(ii) Summary of his working history up to the time of the first medical survey, in terms of the periods spent in the main classes of general environment;

(iii) An unbiased estimate, of known accuracy, of his dust exposure between the two surveys.

For the purposes of this analysis it was decided that the working histories could best be represented in terms of the period spent in "dusty" occupations. This index of the past hazard was preferred to the more complex representation in terms of the three main classes of general environment discussed above, in view of the comparatively small number of men involved and of the small amount of radiological change reported. In any event it is found (using the methods described in Section 4) that the period spent in "dusty" occupations provides an adequate representation of the past hazard and correlates well with the prevalence of pneumoconiosis at the time of the first medical survey.

For this particular analysis it was decided to express the "measured" component of exposure as a single-dimensional variable, in terms of the product of total number count in the 1-5 micron size range and period of exposure; other measures of hazard could, of course, have been calculated on the basis of the information available, depending on the particular hypothesis to be tested. As a result of the environmental sampling programmes carried out by Pneumoconiosis Field Research a "shift exposure index" is calculated for each sampling shift, in terms of the product of average dust concentration (p.p. ml.*, 1-5 microns) and time at risk (hours). The average exposure index for all shifts assigned to each occupational group is then calculated and a man's exposure in the group is obtained as the product of the "group exposure index" and the period spent in the group (for this purpose measured in months). The man's total exposure between the two surveys is obtained by taking the sum of the individual components corresponding to each of the occupational groups to which he has belonged during this period. The units of exposure used in these calculations are thus p.p. ml. (1-5 microns) × hours × months. For example, a figure of 75,000 exposure units could correspond to a period of 10 months spent in an environment where the duration of the working shift was seven and a half hours and the average concentration was 1,000 p.p. ml. (1-5 microns).

When the readings obtained on the first and second surveys are compared there are nine possible sequences of pairs of readings in terms of crossing the boundaries between categories 0 and 1 and categories 1 and 2: (0, 0), (1, 1), (2 or more, 2 or more) [no change]; (0, 1), (0, 2, or more), (1, 2, or more) [apparent progression]; (1, 0), (2 or more, 0), and (2 or more, 1) [apparent regression]. Any apparent regressions are assumed to be due to variations in taking or reading the films and due account is taken of them in the analysis. Although the possibility of true regression of simple pneumoconiosis cannot be entirely ruled out it is considered to be unlikely on the evidence available (Cochrane, 1960). Using the methods which have been described the probability that any of these pairs of readings is obtained may be expressed as a function of the two multi-dimensional dosage variables.

The 485 men considered in the analysis were divided into about 50 different groups, each containing men with similar periods in dusty occupations before the first survey and "measured" exposures. In each group the numbers of men corresponding

*Particles per millilitre.

to the nine pairs of first and second survey readings were recorded. There was a considerable amount of movement from category to category between the two surveys, although the overall progression was small. If all readings of category 2 or more are taken together (as they are in the analysis), a total of 57 men was assigned to a higher category on the second survey than on the first (apparent progression) and 43 men were assigned to a lower category (apparent regression). In view of the relatively high initial prevalence and the small overall change in radiological classification between the two medical surveys, it is apparent on general grounds that the "measured" exposure accounts for only a small proportion of the total hazard experienced by the men concerned. Hence, the accuracy of the resulting estimates of the relation between abnormality and "measured" exposure will be expected to be low.

On the basis of the numbers of men in the various groups assigned to categories 0, 1, and 2 or more on the two surveys and the corresponding periods spent in dusty occupations and "measured" exposures, an estimate was obtained of the abnormality–exposure relation associated with the "measured" exposure separately. [The basic mathematical procedures have been described in detail elsewhere (Ashford *et al.*, 1960)]. Because of the small amount of radiological change certain simplifying assumptions were made in this particular analysis, as the complete (and more time consuming) treatment of the data would not have been worthwhile. The resulting quantal response lines for "measured" exposure corresponding to category 1 or more and category 2 or more pneumoconiosis are shown in Fig. 7a, and the corresponding quantitative abnormality–exposure relation appears at Fig. 7b. The E.D.50 for category 1 or more pneumoconiosis was found to be 72,000 exposure units [p.p. ml. (1-5 microns) × hours × months]. This means, for example, that if a group of men were to work a 7·2 hour shift in an average concentration of 100 p.p. ml. (1-5 microns) at the particular colliery for a period of 100 months they would have a 50% chance of developing category 1 or more pneumoconiosis. As the "measured" exposure is calculated in terms of the product of average concentration and time, this latter remark would apply equally well to a group of men working a 7·2 hour shift in an average concentration of 1,000 p.p. ml. for a period of 10 months, and similarly for any other combination of shift length, average concentration, and period of exposure of which the product comes to 72,000. The E.D. 50 for category 2 or more pneumoconiosis was found to be 135,000 exposure units. It must be emphasized that no quantitative significance should be attached to these figures, as the accuracy of the estimates is poor because of the small amount of progression recorded.

The "fit" of the observations to the underlying assumptions on which the analysis is based was examined by comparing the observed and expected numbers of men showing the various types of response on the two surveys. The agreement was found to be good, and there was no evidence of any significant deviations. In this instance, of course, the amount of progression was small and little information could be obtained about whether or not the methods of analysis had dealt adequately with the "measured" component of exposure. However, the analysis provides a satisfactory example of the potential applications of the method.

This particular study was based on independent readings of the first and second survey films. By a suitable modification of the methods of calculation (Ashford *et al.*, 1960) the same basic treatment may be applied to cover the situation in which the corresponding first and second survey films are classified together.

Discussion

The underlying object of each of the various procedures described above is to determine the direct relation between the radiological abnormality associated with simple pneumoconiosis and some single measure of past hazard. On the basis of this information it is possible to predict, with known accuracy, the abnormality value associated with any given exposure. By comparing the relationships associated with various alternative measures of exposure it will also be possible to determine which provides the most appropriate representation of the past hazard. In this way the main objective of the Pneumoconiosis Field Research, which is to determine safe levels of dustiness, will be achieved. In view of the uncertainties about the relation between radiological pneumoconiosis and disability and about the natural history of P.M.F. which exist at the present time, it would be inappropriate to attempt to lay down any specific criteria for safe levels at this stage. Various investigations which are currently in progress, both within the Pneumoconiosis Field Research and elsewhere, should help to resolve these uncertainties.

The methods of analysis which have been described are based on what is essentially a very simple representation of the relation between abnormality and exposure. The precise mechanisms involved in the development of simple pneumoconiosis may well be extremely complex, but it is assumed that an approach of this kind will provide an adequate description of the various processes involved.

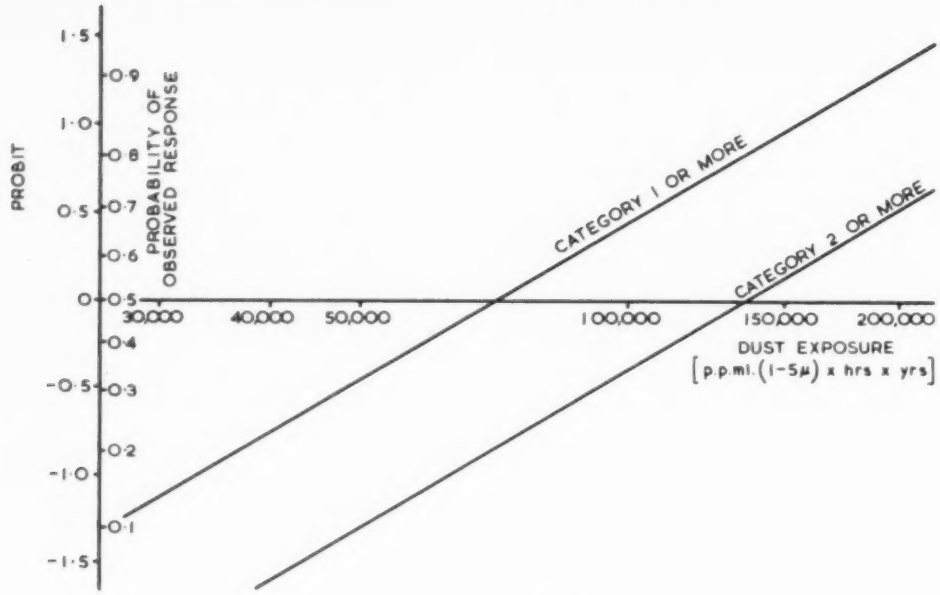

FIG. 7a

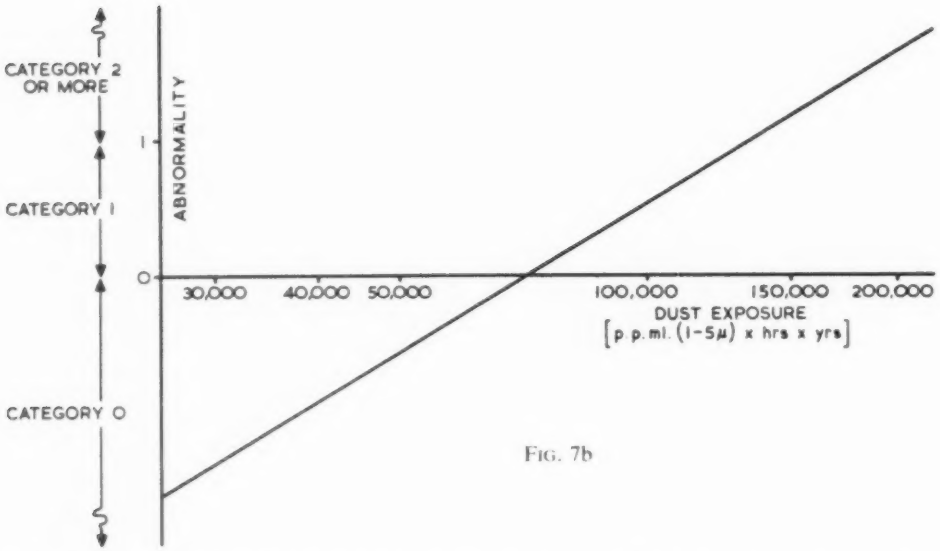

FIG. 7b

FIG. 7. Progression analysis based on the period spent in "dusty" occupations before the first survey and "measured" dust exposure between first and second surveys. (a) The quantal response; (b) The quantitative response.

Within the basic framework of the model it will, however, be possible to test a very wide variety of alternative hypotheses about the nature of the hazard, by varying the basis for calculating the "measured" component of exposure in the progression analyses. In this respect it is perhaps unfortunate that no single hypothesis has yet emerged from all the basic research which has been undertaken into simple pneumoconiosis. It is desirable that any hypothesis which might be put forward as

a result of these statistical analyses should be tested if possible by more fundamental studies before it is adopted as a basis for the control of dust levels.

In each separate analysis it is possible to check that the observed data are consistent with the various assumptions on which the treatment is based. A large number of analyses have been carried out in terms of the relation between the prevalence of pneumoconiosis at the time of the first medical surveys and the past working histories, when measured both as single- and multi-dimensional variables. The observed prevalences of pneumoconiosis have been compared with the corresponding values which would be expected if the model were exact. The frequency of occurrence of significant discrepancies between corresponding figures are very low, and certainly no greater than would have been expected as the result of random variations if the model were exactly true. It is therefore fair to conclude that the underlying assumptions are established, and that they can be applied with confidence in future analyses of the progression data. Nothing can be said about whether some alternative treatment would have led to an even better measure of agreement with the observations. It is, however, possible to assert that if this were the case, the alternative approach must necessarily agree very closely with the methods which have been described here. At this stage in the Research our experience of the progression analyses is limited, but the few results available are consistent with the assumed model. As the methods developed for the progression studies represent a natural extension of those adopted for the analysis of the data from the first round of medical surveys, it seems reasonable to presume that they will be useful in this rather more complicated application.

The use of the retrospective working history data forms an essential part of the methods developed for the progression studies. Although at first sight this approach might appear to be subject to some deficiencies, the analyses of the results of the first medical surveys have established that these working histories can, if expressed in a suitable form, provide a valid measure of past hazard. Since the period before the first medical surveys will account for the greater part of the total hazard of the majority of the men under examination for some years to come, there is an obvious need to take account of this contribution to the man's radiological abnormality.

The study of working populations to determine the relation between pneumoconiosis and past environment is subject to the criticism that they are survivor populations; any tendency for the presence or absence of pneumoconiosis to have an effect on whether a man joins or leaves the population will influence the observed results. However, the effect of simple pneumoconiosis, particularly in its early stages, on respiratory function is known to be small (Carpenter, Cochrane, Gilson, and Higgins, 1956; Rogan *et al.*, 1961) and it is therefore probable that the selective withdrawal of men with minimal or even moderate simple pneumoconiosis will be negligible. This is confirmed by the studies of the dosage–response relationships based on the results of the first medical surveys. For these reasons it is thought that the methods of analysis described in this paper will not be affected appreciably by population selection. On the other hand, P.M.F. is known to be disabling and associated with a reduced expectation of life (Cochrane *et al.*, 1956); it is therefore to be expected that studies of working populations will not provide a valid basis for the assessment of the natural history of complicated pneumoconiosis.

Finally, it must be pointed out that direct comparisons between dosage–response relationships based on two different populations of men involve the tacit assumption that both populations may be regarded as being selected at random from a single larger population. In other words, it must be assumed that there is no difference in the response pattern of the individual populations. Conclusive evidence in support of this assumption is difficult to obtain but on general grounds it seems likely to be justifiable and no evidence to the contrary has been found in any of the analyses.

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THE APRIL (1961) ISSUE

The April (1961) issue contains the following papers:—

"Sooty Manchester" and the Social-Reform Novel 1845-1855. Arthur Pollard.

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Carbon Pneumoconiosis. A. A. Miller and F. Ramsden.

Carbon Pneumoconiosis in a Synthetic Graphite Worker. W. B. Lister.

Byssinosis Among Cotton Workers in Belgium. E. Tuypens.

The Role of Bacterial Endotoxins in Occupational Diseases Caused by Inhaling Vegetable Dusts. B. Pernis, E. C. Vigliani, C. Cavagna, and M. Finulli.

Histamine Liberation by Cotton Dust Extracts: Evidence Against its Causation by Bacterial Endotoxins. H. Antweiler.

Chronic Pulmonary Berylliosis in a Female Chemist. R. I. McCallum, I. Rannie, and C. Verity.

Observations on the Pathogenesis of Silicosis by Means of the Diffusion Chamber Technique. A. G. Heppleston, K. A. Ahlquist, and D. Williams.

The Effects of Vole Bacillus Vaccination of African Mine Workers in the Northern Rhodesian Copper Mines. R. Paul.

Cerebral Tumour in a Lead Worker. R. W. Portal.

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Book Reviews

A number of copies are still available and may be obtained from the Publishing Manager, British Medical Association, Tavistock Square, W.C.1, price 17*s.* 6*d.*

Brit. J. industr. Med., 1961, **18**, 197.

SICKNESS ABSENCE OF WOMEN BUS CONDUCTORS IN LONDON TRANSPORT (1953-1957)

BY

C. J. CORNWALL and P. A. B. RAFFLE

From the London Transport Executive

(RECEIVED FOR PUBLICATION MARCH 27, 1961)

The sickness-absence experience of women bus conductors employed by London Transport on their central (red) buses is analysed for the years 1953 to 1957 inclusive. The experience of single and married women is given separately, and only absences lasting four days or more (all of which are supported by medical certificate) are included. Results are given in quinquennial age groups for each of 20 broad diagnostic groups and comment is made on each group, emphasis being placed on the comparative experiences of single and married women. Both the numbers of spells of absence and the numbers of days of absence are considered. It is found generally that married women conductors have considerably more sickness absence than single women conductors of the same age: this applies both to spells and days of absence. Analysis of the difference by diagnostic group suggests that there is a propensity, of social origin, for married women to have more absences.

An account of the principles and methods followed in the compilation of the London Transport sickness-absence statistics was given in *Health in Industry* (London Transport Executive, 1956). The sickness-absence experience of various groups of London Transport male wages staff (including male conductors) and male and female clerical staff over the period 1949-52 was set out in detail in that publication, analysed by age and by broad diagnostic group. This paper extends that work by giving some similar analyses, but for the subsequent quinquennium, for a group of London Transport female wages staff, namely the women conductors of the central (red) buses.

The sickness-absence experience of single and married women has been separated, and emphasis has been placed on the comparison of the sickness absence of these "single" and "married" women who both follow the same occupation. The subdivision into single and married women is according to whether they are known as "Miss" or "Mrs" respectively; information is not available to the employer enabling the "Mrs" group to be subdivided into women with husbands living and widows.

Recruitment of women conductors started in 1940, was discontinued after the war, recommenced in 1950, and has continued to the present time. Thus the population under consideration consists mainly of women with less than eight years' service but there are some with up to 18 years' service.

"Sickness absence" is taken to mean absence from work accepted by the employer as attributable to sickness or injury. Medical certificates are required not later than the third day of absence. This investigation has been limited to spells of absence lasting four days or longer, and thus includes only absences supported by a medical certificate. The majority of spells of sickness absence terminate with the employee's recovery and return to her former work. When an absence lasts for six months, however, the recorded absence is curtailed at 182 days, as after that duration of absence, an administrative decision may be taken regarding the retention of the employee's name on the books, or her premature retirement on the grounds of ill health. It should be noted that in all cases, the number of days' absence ascribed to a particular spell of absence is the number of days on the calendar from the first day of absence from work to the day preceding the employee's return to work, both dates inclusive; in other words, the length of a spell of absence in days is recorded on the basis of seven days to the week.

Women conductors are medically examined before appointment and must reach appropriate physical standards. An employee who has been absent for more than four weeks (or in the case of certain

diagnoses certified by the general practitioner, including accidents, for a shorter period) must be certified fit by a London Transport Medical Officer before resuming duty. When the appropriate standard of health is not reached, the employee may be found suitable alternative employment, but the scope for alternative employment of women in the London Transport Executive's undertaking is very limited.

This investigation covers the five years 1953 to 1957. Sick pay was not paid to women conductors before December 1956. Since then, limited amounts have been payable, after a week of absence, to women conductors who are over age 25 and who have more than five years' service. Many of the married women do not contribute to the National Insurance Scheme, and thus receive no sickness benefit from the State. Women conductors have been eligible for membership of the Sick Benefit Section of a large Friendly Society, which draws its membership mainly from the bus crews, only since March 1957. Thus throughout the majority of the quinquennium under review, women could not obtain sickness benefit from this source.

The detailed results of the investigation are set out in Tables 1 to 21 in the Appendix. In these Tables data are given for single and married women, and subdivided into quinquennial age groups. Sickness absence has been split into 20 broad diagnostic groups, which conform to those given by London Transport Executive (1956), and are based on the three-figure International Statistical Classification of Diseases and Injuries adopted in 1948 by the World Health Organization.

For each diagnostic group, and each quinquennial age group, the following indices have been calculated:

(i) *Average annual duration per person*, which is the average number of calendar days of sickness absence per employee per annum;
(ii) *Annual inception rate (spells)*, which is the average number of spells commencing in a year per employee;
(iii) *Average length of spell*, which is the average length in days of each recorded spell of sickness absence.

In addition, the number of "man-years" of exposure to risk, the number of spells of absence commencing in the period, and the number of days of absence during the period are given. It is emphasized that these only relate to spells of four days' duration or more.

Graphs have been drawn to assist in the interpretation of the Tables, with particular reference to the comparison between the sickness-absence experiences of married and single women. There were no single women bus conductors older than 60, the normal retiring age, and very few married women conductors over that age; the graphs have therefore been curtailed at age 60. It should also be noted that the exposed-to-risk figures for single women in the 50-54 and the 55-59 age groups and for married women in the 55-59 age group are very small, and consequently little weight should be attached to the results for those groups. The corresponding sections of the graphs are shown as dotted lines.

Broad Diagnostic Groups

All Diagnostic Groups Combined.—(Table 1).

Graph 1a and b show quite clearly that the single women bus conductors experience significantly lighter sickness absence than the married women. This is so at all ages. The excess for married women is of the order of 40% for spells, and as much as 60% for days of absence. In each case, the annual inception rate (spells) remains fairly constant, but the average annual duration per person increases steadily with age.

BDG. 1.—Tuberculosis (Table 2) (International Code Nos. 001-019).

There were only seven spells among the single women, compared with 11 amongst the married women. This difference is not significant. No comparison by age is possible with such small numbers. The curtailment of absence at 182 days has a particularly important effect on the recording of absences caused by tuberculosis.

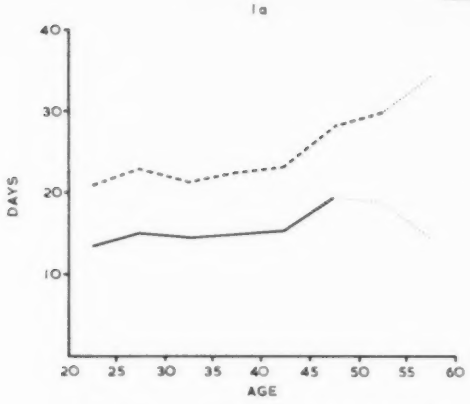
BDG. 2.—Infective and Parasitic Diseases (Table 3) (International Code Nos. 020-138).

Although the rates are rather erratic, there is a general tendency for single women to have the better experience (Graph 2a and b). The low rates, both for spells and days, in the age-group 35-44, are probably due to the termination of childhood infective diseases. After age 45, the only disease of any numerical importance is herpes zoster. It may be that single women have the better experience because they have less contact with children.

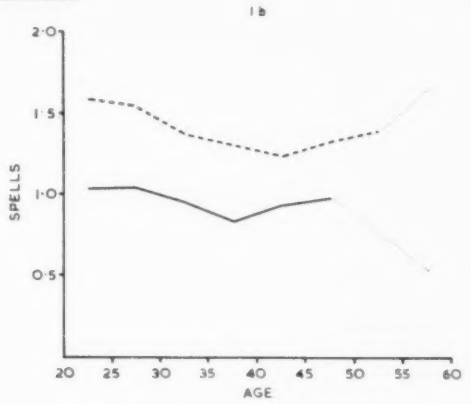
BDG. 3.—Neoplasms (Table 4) (International Code Nos. 140-239).

In nearly every age group, single women had the lower incidence of spells. The effect on days of absence was not so marked. There was a definite increase in the average annual duration per person as age increased (Graph 3a and b): this is the same trend as for male conductors given in *Health in Industry* (1956), but is not so marked at ages over 50.

ALL DIAGNOSES



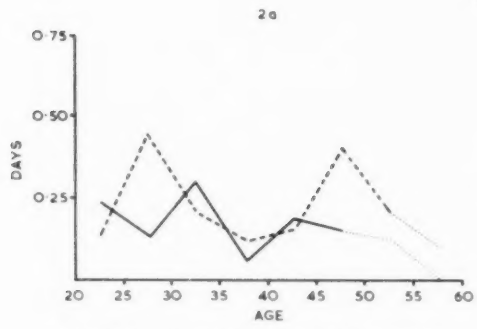
GRAPH 1a.—Average annual duration per person.



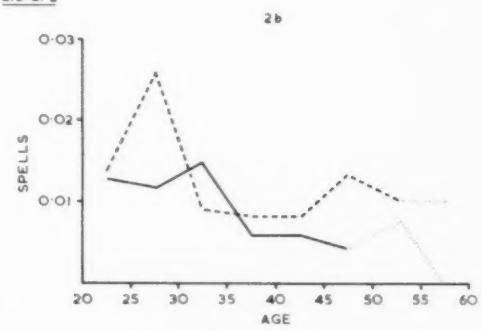
GRAPH 1b.—Annual inception rate (spells).

Key: ———— Single women (..... over age 50); - - - - - - Married women (..... over age 55).

B.D.G. 2



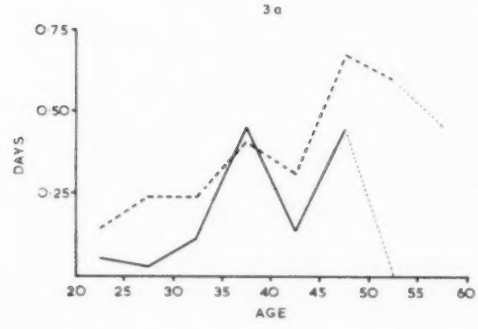
GRAPH 2a.—Average annual duration per person.



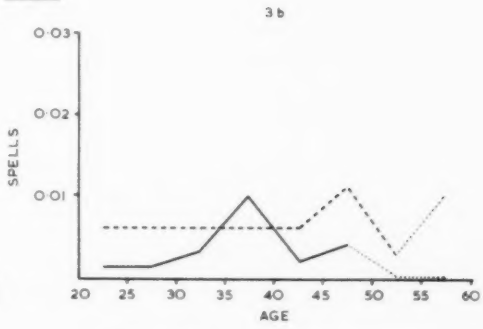
GRAPH 2b.—Annual inception rate (spells).

Key: ———— Single women ..... over age 50); - - - - - - Married women (..... over age 55).

B.D.G. 3

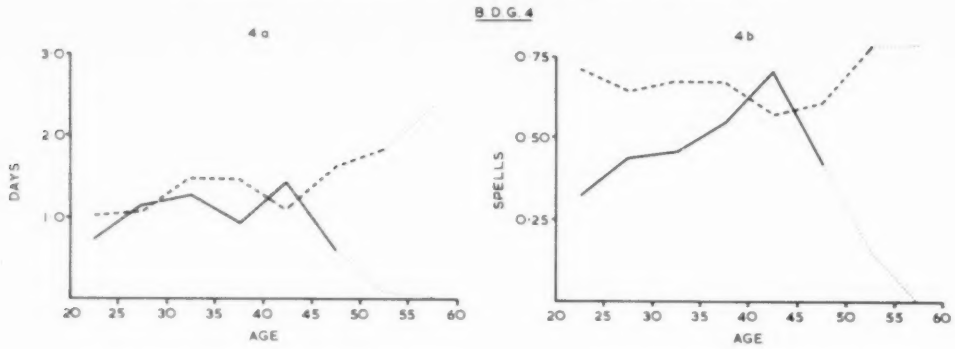


GRAPH 3a.—Average annual duration per person.

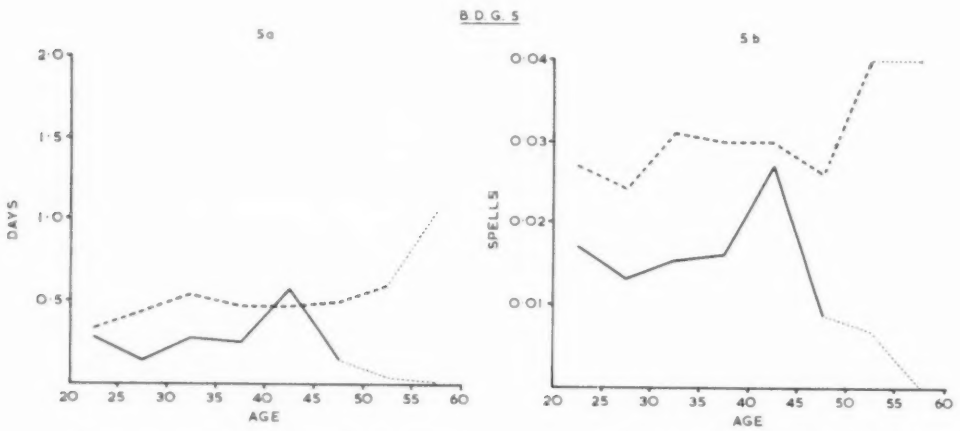


GRAPH 3b.—Annual inception rate (spells).

Key: ———— Single women (..... over age 50); - - - - - - Married women (..... over age 55).



GRAPH 4a.—Average annual duration per person.
GRAPH 4b.—Annual inception rate (spells).
Key: ——— Single women (..... over age 50); - - - - - - Married women (..... over age 55).



GRAPH 5a.—Average annual duration per person.
GRAPH 5b.—Annual inception rate (spells).
Key: ——— Single women (..... over age 50); - - - - - - Married women (..... over age 55).

BDG. 4.—Functional Nervous Disorders (Table 5) (International Code Nos. 300-318).

The annual inception rate (spells) is lower for single women at nearly all ages, but there is little difference between the sickness-absence experience of married and single women as far as the average annual durations per person are concerned. The rise in rates with age at the younger ages of single women is considerable and not unexpected (Graph 4a and b).

BDG. 5.—Organic Nervous Disorders (Table 6) (International Code Nos. 330-369, 780, 781).

In nearly every age group, the single women's experience is the lower. This is particularly marked after age 45, when the single women's rates decrease as age increases, but it must be borne in mind that the number of single women bus conductors over age 50 is not very large (Graph 5a and b).

BDG. 6.—Diseases of the Eye (Table 7) (International Code Nos. 370-389).

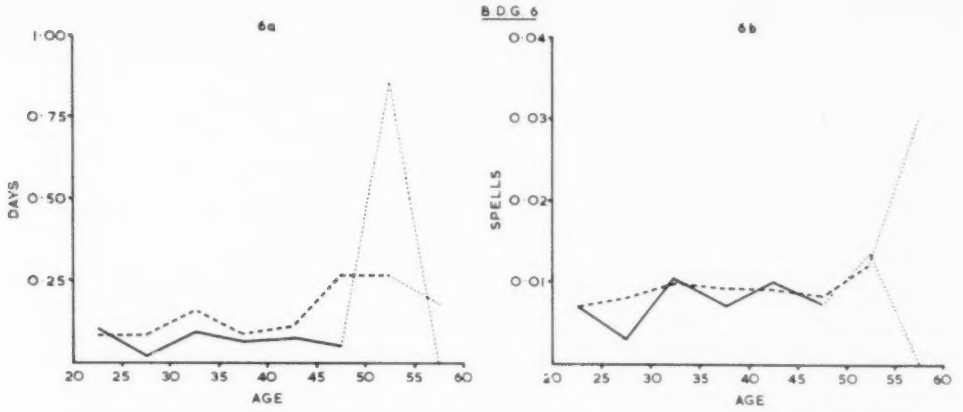
In this diagnostic group, the rates for married and single women are similar (Graph 6a and b).

BDG. 7.—Diseases of the Ear (Table 8) (International Code Nos. 390-398).

As in diseases of the eye, there is little difference between the rates for married and single women. The diagnoses included in groups 6 and 7 are usually precise, and in neither group is there any reason to expect any differences in incidence between married and single women (Graph 7a and b).

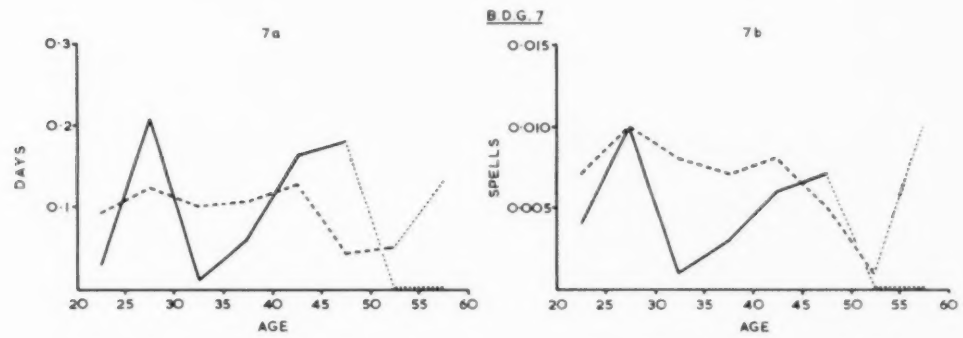
BDG. 8.—Diseases of the Circulatory System (Table 9) International Code Nos. 400-468, 782).

Both rates are consistently lower for single women (Graph 8a and b). There is a considerable increase in the rates for married women after age 45.



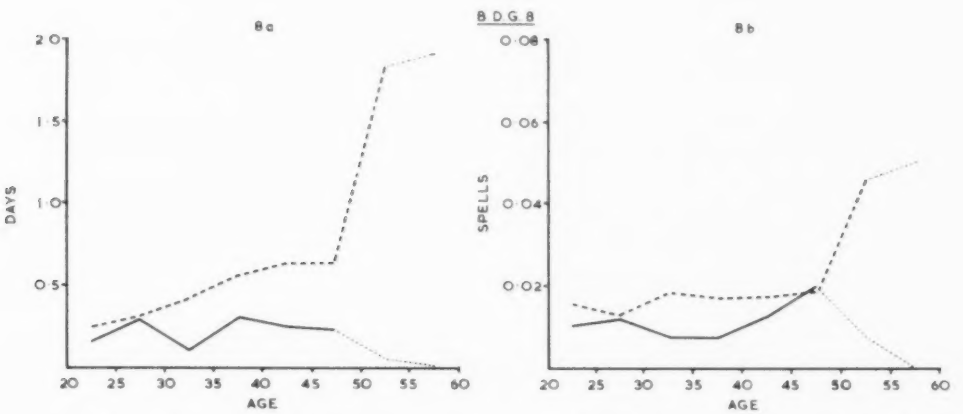
GRAPH 6a.—Average annual duration per person.
Key: ———— Single women (..... over age 50); - - - - - - Married women (..... over age 55).

GRAPH 6b.—Annual inception rate (spells).



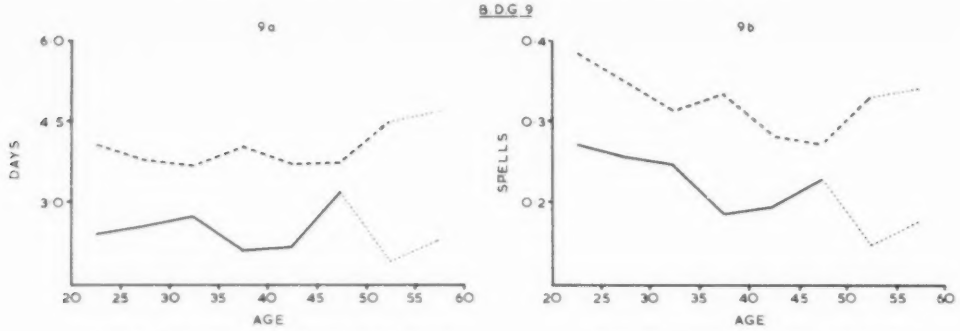
GRAPH 7a.—Average annual duration per person.
Key: ———— Single women (..... over age 50); - - - - - - Married women (..... over age 55).

GRAPH 7b.—Annual inception rate (spells).



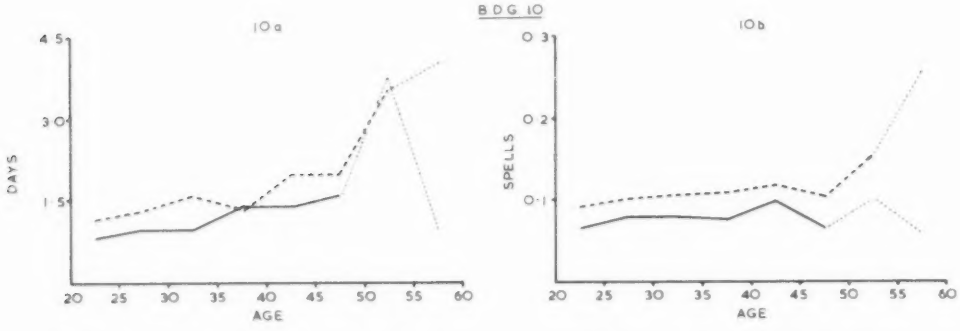
GRAPH 8a.—Average annual duration per person.
Key: ———— Single women (..... over age 50); - - - - - - Married women (..... over age 55).

GRAPH 8b.—Annual inception rate (spells).



GRAPH 9a.—Average annual duration per person.
Key: ———— Single women (. . . . . over age 50); – – – – – Married women (. . . . . over age 55).

GRAPH 9b.—Annual inception rate (spells).
Key: ———— Single women (. . . . . over age 50); – – – – – Married women (. . . . . over age 55).



GRAPH 10a.—Average annual duration per person.
Key: ———— Single women (. . . . . over age 50); – – – – – Married women (. . . . . over age 55).

GRAPH 10b.—Annual inception rate (spells).
Key: ———— Single women (. . . . . over age 50); – – – – – Married women (. . . . . over age 55).

Varicose veins are included in this group and are known to be more common among married women.

BDG. 9.—Colds and Influenza (Table 10) (International Code Nos. 470, 480-483).

This group accounts for about one-quarter of the spells of more than three days' duration of single and married women. Rates are, however, considerably lower for single women than for married women, in some age groups by as much as 50% for spells and over 100% for days per person (Graph 9a and b). It will be remembered that colds and influenza are thought to be more common in families with school children.

BDG. 10.—Bronchitis (Table 11) (International Code Nos. 500-502).

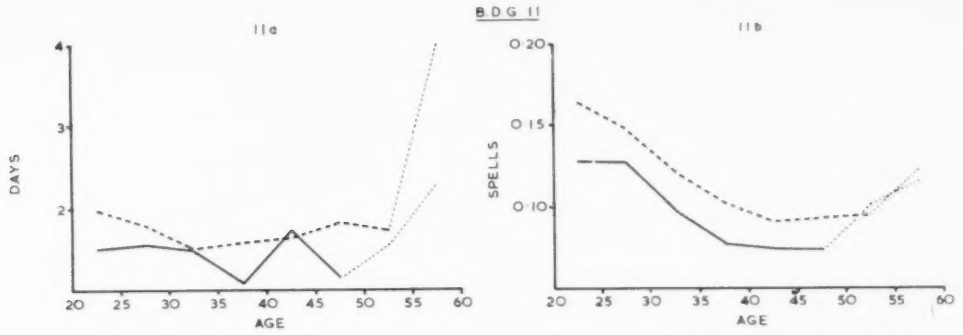
The annual inception rate (spells) for single women is consistently lower than for married women, but the differences in days of absence are not so large (Graph 10a and b). As might be expected, the average length of spell increases with age. It has not been possible to investigate geographical variations in this paper. Our recent paper on bronchitis (Cornwall and Raffle, 1961) discusses this point. Differences in the smoking habits of single and married women may account for the differences noted, but there is no evidence to support this.

BDG. 11.—Other Respiratory Diseases (Table 12) (International Code Nos. 471-475, 490-493, 510-527, 763, 783).

This is a heterogeneous group, and no firm conclusions are possible. Although the annual inception rate (spells) for single women is consistently lower at ages up to 50, the percentage excess for married women is only about 20%. The pattern is less clear for average annual duration (Graph 11a and b). The incidence of spells decreases as age increases, up to age 50. There was a similar trend for male bus conductors for the previous quinquennium, given in *Health in Industry* (1956).

BDG. 12.—Diseases of the Stomach and Duodenum (Table 13) (International Code Nos. 540-545, 784).

No clear pattern emerges here (Graph 12a and b). There is no increase in the incidence of this type of



GRAPH 11a.—Average annual duration per person.
Key: ——— Single women (..... over age 50); – – – – – Married women (..... over age 55).

GRAPH 11b.—Annual inception rate (spells).

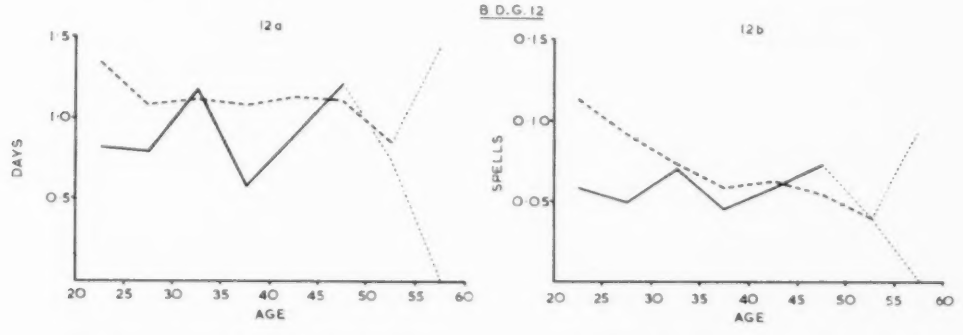
disease as age increases. This is in marked contrast to the results for men given in *Health in Industry* (1956), and referred to in detail by Raffle (1959).

BDG. 13.—Hernia of Abdominal Cavity (Table 14) (International Code Nos. 560-561).

There are very few data in this diagnostic group.

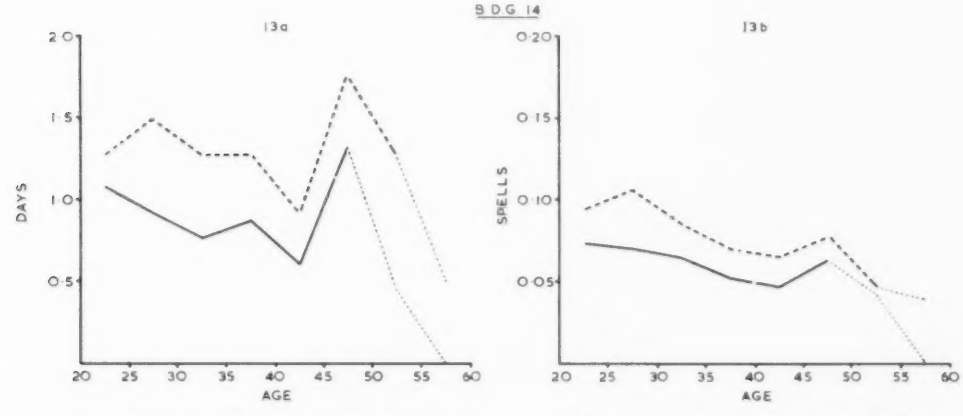
BDG. 14.—Other Diseases of the Digestive System (Table 15) (International Code Nos. 530-539, 550-553, 570-587, 764, 785).

This is a heterogeneous group, but at all ages,



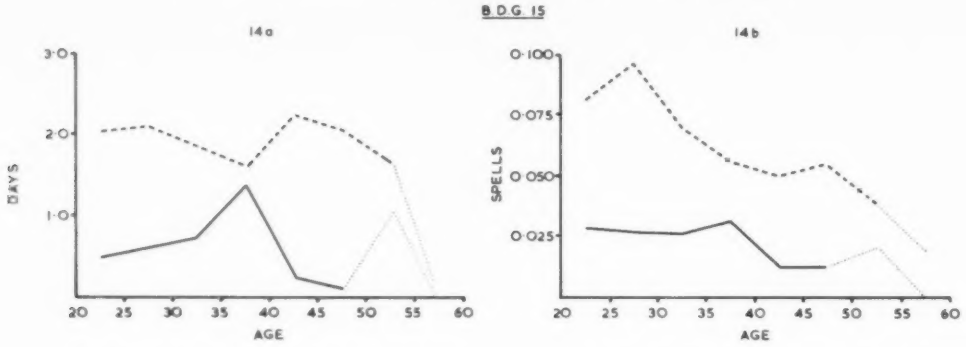
GRAPH 12a.—Average annual duration per person.
Key: ——— Single women (..... over age 50); – – – – – Married women (..... over age 55).

GRAPH 12b.—Annual inception rate (spells).



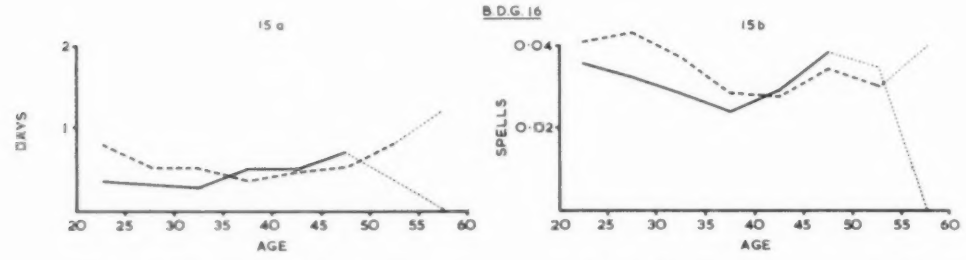
GRAPH 13a.—Average annual duration per person.
Key: ——— Single women (..... over age 50); – – – – – Married women (..... over age 55).

GRAPH 13b.—Annual inception rate (spells).



GRAPH 14a.—Average annual duration per person.
Key: ——— Single women (..... over age 50); – – – – – Married women (..... over age 55).

GRAPH 14b.—Annual inception rate (spells).



GRAPH 15a.—Average annual duration per person.
Key: ——— Single women (..... over age 50); – – – – – Married women (..... over age 55).

GRAPH 15b.—Annual inception rate (spells).

married women's rates are higher (Graph 13a and b) than those of single women. The excess is of the order of 30% for spells and 35% for days.

In contrast to the experience of male bus conductors, given in *Health in Industry* (1956), the incidence of spells of sickness absence attributed to this diagnostic group decreases as age increases, both for married and single women conductors.

BDG. 15.—Diseases of Women (Table 16) (International Code Nos. 620-689).

At all ages, single women's rates are lower, by an appreciable margin, as would be expected (Graph 14a and b).

BDG. 16.—Diseases of the Skin (Table 17) (International Code Nos. 690-698, 700-716).

This group shows little difference between the rates of married and single women, and little variation with age (Graph 15a and b). If the use of modern detergents is a factor in the incidence of sickness absence in this diagnostic group, it might have been expected that married women's rates would have been the higher.

BDG. 17.—Diseases of Bones and Organs of Movement (Table 18) (International Code Nos. 720-749, 787).

The incidence of sickness absence attributed to this group appears to increase generally with age. For the annual inception rate (spells), single women's rates are consistently lower than those of married women, but for days, the differences are not so marked (Graph 16a and b).

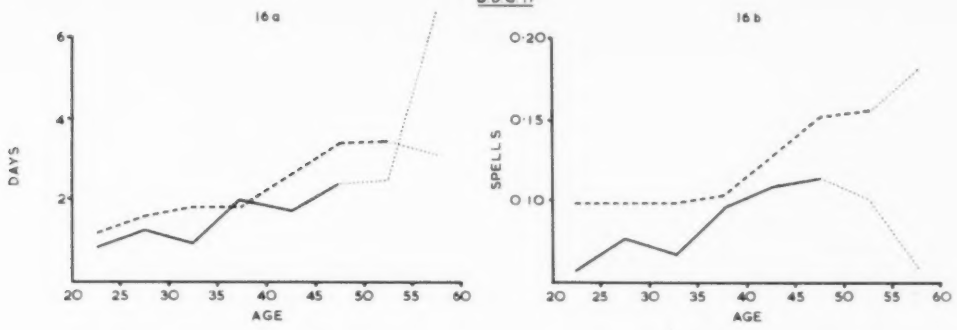
BDG. 18.—Accidents on Duty (Table 19) (International Code Nos. (N) 800-999).

Single women bus conductors experience lower rates of absence caused by accidents on duty. It does not necessarily follow, however, that single women bus conductors have less accidents than married women bus conductors, but merely that they have fewer absences (of more than three days' duration) resulting from accidents (Graph 17a and b).

BDG. 19.—Accidents off Duty (Table 20) (International Code Nos. (N) 800-999).

In this group again, the single women's rates are predominantly lower. This should possibly be expected, as most married women undertake the dual task of conducting a bus and running a home.

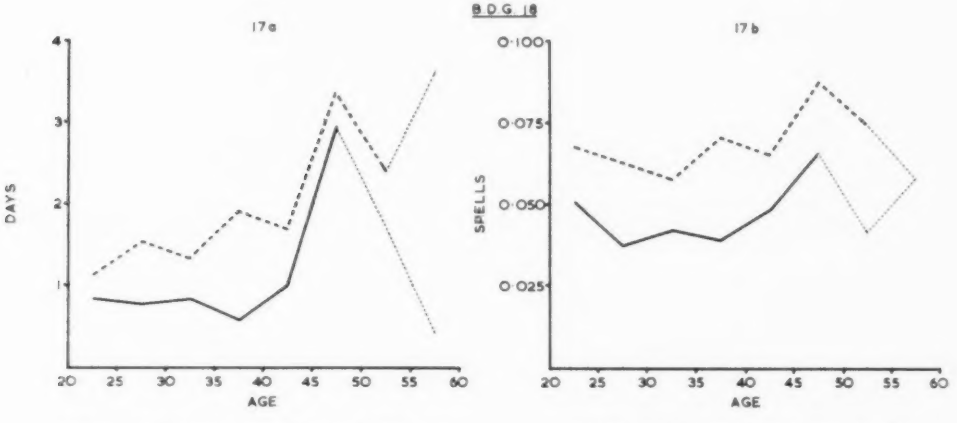
B.D.G. 17



GRAPH 16a.—Average annual duration per person.
Key: ———— Single women (. . . . . over age 50); - - - - - - Married women (. . . . . over age 55).

GRAPH 16b.—Annual inception rate (spells).

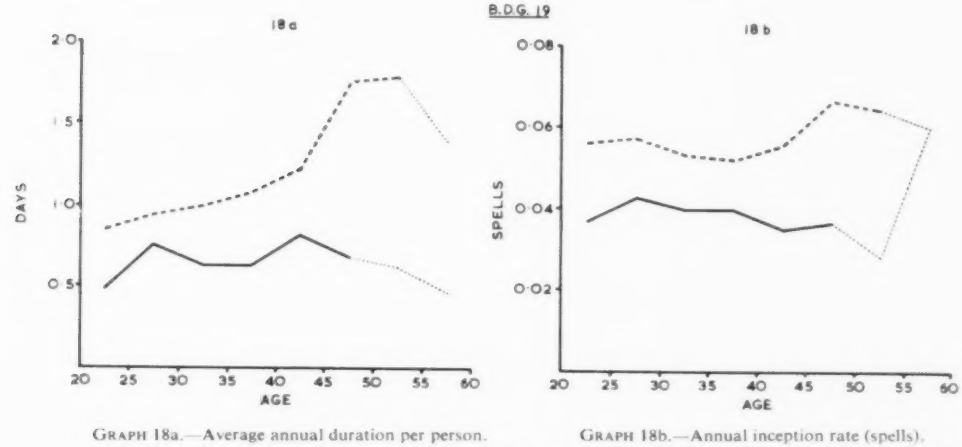
B.D.G. 18



GRAPH 17a.—Average annual duration per person.
Key: ———— Single women (. . . . . over age 50); - - - - - - Married women (. . . . . over age 55).

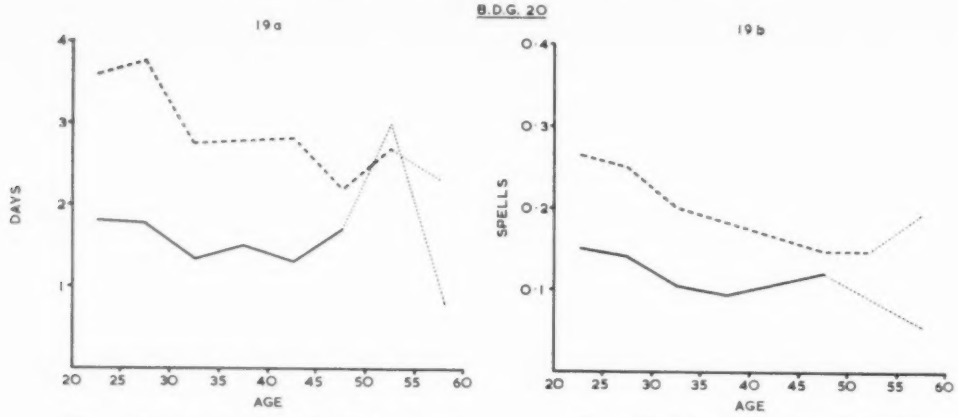
GRAPH 17b.—Annual inception rate (spells).

B.D.G. 19



GRAPH 18a.—Average annual duration per person.
Key: ———— Single women (. . . . . over age 50); - - - - - - Married women (. . . . . over age 55).

GRAPH 18b.—Annual inception rate (spells).



GRAPH 19a.—Average annual duration per person.
Key: ——— Single women (. . . . . over age 50); – – – – – Married women (. . . . . over age 55).

GRAPH 19b.—Annual inception rate (spells).

There is no general increase of spells with age. (See Graph 18a and b.)

BDG. 20.—Miscellaneous (Table 21) (International Code Nos. 000, 240-299, 320-326, 590-617, 750-762, 765-776, 786, 788-795).

The lower rates for single women are probably only a reflection of the general tendency in most diagnostic groups (Graph 19a and b).

Conclusion

It may be concluded from the above analysis that single women bus conductors experience appreciably less sickness absence than their married women colleagues. The extent of the difference for each diagnostic group may best be measured by the ratio of the rate (for spells or days) for all married women to the rate for all single women, the rates being indirectly standardized to allow for the different age distributions of the two groups. The following Table sets out the results.

It will be observed that, for most diagnostic groups, the percentage differences are very similar to the average for all diagnoses. This suggests that there is a propensity, of a social origin, for married women to have more absences: it is spread over all diagnoses and the resulting increase in rates is varied only (a) downwards, in the relatively few diagnostic groups where a condition can be accurately diagnosed, or (b) upwards, where there are sound medical reasons for a difference in actual morbidity.

We wish to express our thanks to Mr. F. H. Spratling, F.I.A., Chief Establishment Officer, and Dr. L. G.

TABLE

PERCENTAGE RATIO OF SICKNESS OF MARRIED WOMEN TO THAT OF SINGLE WOMEN, ADJUSTED FOR AGE

Broad Diagnostic Group No.	Short Descriptions	% Ratio of Average Annual Durations (Days)	% Ratio of Annual Inception Rates (Spells)
1	Tuberculosis	159	117
2	Infective and parasitic diseases	134	132
3	Neoplasms	195	200
4	Functional nervous disorders	124	138
5	Organic nervous disorders	161	166
6	Diseases of the eye	134	117
7	Diseases of the ear	100	143
8	Diseases of the circulatory system	197	149
9	Colds and influenza	144	135
10	Bronchitis	119	133
11	Other respiratory diseases	117	120
12	Diseases of the stomach and duodenum	122	130
13	Hernia of abdominal cavity	367	1,205
14	Other diseases of the digestive system	140	133
15	Diseases of women	280	267
16	Diseases of the skin	129	113
17	Diseases of bones and organs of movement	130	133
18	Accidents on duty	155	139
19	Accidents off duty	155	138
20	Miscellaneous	175	164
	All diagnostic groups	147	140

Norman, Chief Medical Officer, of London Transport, for their help and encouragement in the preparation of this paper, to Mr. J. E. Ager and the staff of the Central Record and Actuarial Sections for the machine work and computation involved, and to Mrs. A. Garey who has drawn the graphs.

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APPENDIX

ABSENCES OF FOUR DAYS DURATION OR MORE IN MARRIED AND SINGLE WOMEN BUS CONDUCTORS, 1953-1957.

TABLE 1: ALL DIAGNOSES

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	27,413	19,467	13·049	21·550	2,151	1,442	1·024	1·596	12·7	13·5
25-29	1,668	1,971	24,513	45,835	14·683	23·238	1,710	3,057	1·024	1·550	14·3	15·0
30-34	1,082	2,447	15,434	52,561	14·261	21·497	1,026	3,355	0·948	1·372	15·0	15·7
35-39	694	2,611	10,160	58,623	14·630	22·453	599	3,449	0·863	1·321	17·0	17·0
40-44	625	2,679	9,637	62,358	15·419	23·260	587	3,318	0·939	1·238	16·4	18·8
45-49	460	1,898	8,889	53,037	19·325	27·950	446	2,468	0·970	1·301	19·9	21·5
50-54	142	670	2,642	20,174	18·671	30·079	111	920	0·784	1·372	23·8	21·9
55-59	17	100	239	3,394	14·059	34·110	10	162	0·588	1·628	23·9	21·0
(60 and over)	—	15	—	665	—	44·334	—	19	—	1·267	—	35·0
Total	6,788	13,295	98,927	316,114	—	—	6,640	18,190	—	—	—	—

TABLE 2: BROAD DIAGNOSTIC GROUP 1: TUBERCULOSIS
(I.S.C. Nos. 001-019)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	260	179	0·124	0·198	3	—	0·001	—	86·7	—
25-29	1,668	1,971	574	1,113	0·344	0·564	4	5	0·002	0·003	143·5	222·6
30-34	1,082	2,447	180	392	0·166	0·160	—	2	—	0·001	—	196·0
35-39	694	2,611	—	326	—	0·125	—	3	—	0·001	—	108·7
40-44	625	2,679	—	—	—	—	—	—	—	—	—	—
45-49	460	1,898	—	182	—	0·096	—	1	—	0·001	—	182·0
50-54	142	670	—	—	—	—	—	—	—	—	—	—
55-59	17	100	—	—	—	—	—	—	—	—	—	—
(60 and over)	—	15	—	—	—	—	—	—	—	—	—	—
Total	6,788	13,295	1,014	2,192	—	—	7	11	—	—	—	—

TABLE 3: BROAD DIAGNOSTIC GROUP 2: INFECTIVE AND PARASITIC DISEASES
(I.S.C. Nos. 020-138)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	500	113	0·238	0·125	28	13	0·013	0·014	17·9	8·7
25-29	1,668	1,971	203	891	0·122	0·452	20	51	0·012	0·026	10·2	17·5
30-34	1,082	2,447	322	499	0·298	0·204	16	22	0·015	0·009	20·1	22·7
35-39	694	2,611	43	278	0·062	0·106	4	21	0·006	0·008	10·8	13·2
40-44	625	2,679	102	390	0·163	0·145	4	22	0·006	0·008	25·5	17·7
45-49	460	1,898	68	752	0·148	0·396	2	26	0·004	0·014	34·0	28·9
50-54	142	670	16	137	0·113	0·204	1	7	0·007	0·010	16·0	19·6
55-59	17	100	—	9	—	0·090	—	1	—	0·010	—	9·0
(60 and over)	—	15	—	—	—	—	—	—	—	—	—	—
Total	6,788	13,295	1,254	3,069	—	—	75	163	—	—	—	—

*Excludes days beyond first six months of absence.

TABLE 4: BROAD DIAGNOSTIC GROUP 3: NEOPLASMS (I.S.C. Nos. 140-239)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	103	134	0·049	0·148	3	5	0·001	0·006	34·3	26·8
25-29	1,668	1,971	29	462	0·017	0·234	1	11	0·001	0·006	29·0	42·0
30-34	1,082	2,447	110	558	0·102	0·228	3	14	0·003	0·006	36·7	39·9
35-39	694	2,611	309	1,098	0·445	0·421	7	15	0·010	0·006	44·1	73·2
40-44	625	2,679	82	831	0·131	0·310	1	16	0·002	0·006	82·0	51·9
45-49	460	1,898	200	1,265	0·435	0·667	2	21	0·004	0·011	100·0	60·2
50-54	142	670	—	396	—	0·590	—	2	—	0·003	—	198·0
55-59	17	100	—	44	—	0·442	—	1	—	0·010	—	44·0
(60 and over)	—	15	—	47	—	3·133	—	—	—	—	—	—
Total	6,788	13,295	833	4,835	—	—	17	85	—	—	—	—

TABLE 5: BROAD DIAGNOSTIC GROUP 4: FUNCTIONAL NERVOUS DISORDERS (I.S.C. Nos. 300-318)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	1,312	945	0·625	1·046	70	63	0·033	0·070	18·7	15·0
25-29	1,668	1,971	1,957	2,167	1·172	1·099	71	127	0·043	0·064	27·6	17·1
30-34	1,082	2,447	1,433	3,608	1·324	1·476	49	164	0·045	0·067	29·2	22·0
35-39	694	2,611	620	3,975	0·893	1·522	39	176	0·056	0·067	15·9	22·6
40-44	625	2,679	878	2,938	1·405	1·096	43	154	0·069	0·057	20·4	19·1
45-49	460	1,898	287	2,982	0·624	1·572	18	113	0·039	0·060	15·9	26·4
50-54	142	670	16	1,193	0·113	1·779	2	53	0·014	0·079	8·0	22·5
55-59	17	100	—	235	—	2·362	—	8	—	0·080	—	29·4
(60 and over)	—	15	—	53	—	3·533	—	1	—	0·067	—	53·0
Total	6,788	13,295	6,503	18,096	—	—	292	859	—	—	—	—

TABLE 6: BROAD DIAGNOSTIC GROUP 5: ORGANIC NERVOUS DISORDERS (I.S.C. Nos. 330-369, 780, 781)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	613	297	0·292	0·329	36	24	0·017	0·027	17·0	12·4
25-29	1,668	1,971	237	814	0·142	0·413	22	47	0·013	0·024	10·8	17·3
30-34	1,082	2,447	297	1,335	0·274	0·546	16	77	0·015	0·031	18·6	17·3
35-39	694	2,611	169	1,207	0·243	0·462	11	79	0·016	0·030	15·4	15·3
40-44	625	2,679	359	1,216	0·574	0·454	17	80	0·027	0·030	21·1	15·2
45-49	460	1,898	62	892	0·135	0·470	4	49	0·009	0·026	15·5	18·2
50-54	142	670	6	389	0·042	0·580	1	27	0·007	0·040	6·0	14·4
55-59	17	100	—	136	—	1·367	—	4	—	0·040	—	34·0
(60 and over)	—	15	—	—	—	—	—	—	—	—	—	—
Total	6,788	13,295	1,743	6,286	—	—	107	387	—	—	—	—

TABLE 7: BROAD DIAGNOSTIC GROUP 6: DISEASES OF THE EYE (I.S.C. Nos. 370-389)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	231	82	0·110	0·091	14	6	0·007	0·007	16·5	13·7
25-29	1,668	1,971	44	181	0·026	0·092	5	16	0·003	0·008	8·8	11·3
30-34	1,082	2,447	107	420	0·099	0·172	12	25	0·011	0·010	8·9	16·8
35-39	694	2,611	42	184	0·060	0·070	5	23	0·007	0·009	8·4	8·0
40-44	625	2,679	43	301	0·069	0·112	6	25	0·010	0·009	7·2	12·0
45-49	460	1,898	20	502	0·043	0·265	3	16	0·007	0·008	6·7	31·4
50-54	142	670	121	179	0·855	0·267	2	8	0·014	0·012	60·5	22·4
55-59	17	100	—	18	—	0·181	—	3	—	0·030	—	6·0
(60 and over)	—	15	—	—	—	—	—	—	—	—	—	—
Total	6,788	13,295	608	1,867	—	—	47	122	—	—	—	—

*Excludes days beyond first six months of absence.

TABLE 8: BROAD DIAGNOSTIC GROUP 7: DISEASES OF THE EAR (I.S.C. Nos. 390-398)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	63	81	0·030	0·090	9	6	0·004	0·007	7·0	13·5
25-29	1,668	1,971	353	251	0·211	0·127	16	19	0·010	0·010	22·0	13·2
30-34	1,082	2,447	12	252	0·011	0·103	1	20	0·001	0·008	12·0	12·6
35-39	694	2,611	45	276	0·065	0·106	2	19	0·003	0·007	22·5	14·5
40-44	625	2,679	97	354	0·155	0·132	4	21	0·006	0·008	24·3	16·9
45-49	460	1,898	84	85	0·183	0·045	3	9	0·007	0·005	28·0	9·4
50-54	142	670	—	36	—	0·054	—	1	—	0·001	—	36·0
55-59	17	100	—	13	—	0·131	—	1	—	0·010	—	13·0
(60 and over)	—	15	—	—	—	—	—	—	—	—	—	—
Total	6,788	13,295	653	1,348	—	—	35	96	—	—	—	—

TABLE 9: BROAD DIAGNOSTIC GROUP 8: DISEASES OF THE CIRCULATORY SYSTEM (I.S.C. Nos. 400-468, 782)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	334	190	0·159	0·210	20	14	0·010	0·015	16·7	13·6
25-29	1,668	1,971	461	605	0·276	0·307	21	25	0·013	0·013	22·0	24·2
30-34	1,082	2,447	107	1,021	0·099	0·418	8	46	0·007	0·019	13·4	22·2
35-39	694	2,611	217	1,423	0·312	0·545	5	44	0·007	0·017	43·4	32·3
40-44	625	2,679	162	1,663	0·259	0·620	8	45	0·013	0·017	20·3	37·0
45-49	460	1,898	100	1,162	0·217	0·612	9	36	0·020	0·019	11·1	32·3
50-54	142	670	11	1,224	0·078	1·835	1	30	0·007	0·045	11·0	40·8
55-59	17	100	—	188	—	1·889	—	5	—	0·050	—	37·6
(60 and over)	—	15	—	—	—	—	—	—	—	—	—	—
Total	6,788	13,295	1,392	7,476	—	—	72	245	—	—	—	—

TABLE 10: BROAD DIAGNOSTIC GROUP 9: COLDS AND INFLUENZA (I.S.C. Nos. 470, 480-483)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	5,066	3,599	2·411	3·984	559	347	0·266	0·384	9·1	10·4
25-29	1,668	1,971	4,297	7,373	2·574	3·738	430	682	0·258	0·346	10·0	10·8
30-34	1,082	2,447	2,971	8,974	2·745	3·670	262	760	0·242	0·311	11·3	11·8
35-39	694	2,611	1,457	10,944	2·098	4·192	129	893	0·186	0·342	11·3	12·3
40-44	625	2,679	1,424	9,771	2·278	3·645	121	752	0·194	0·280	11·8	13·0
45-49	460	1,898	1,458	6,963	3·170	3·670	105	513	0·228	0·270	13·9	13·6
50-54	142	670	270	3,004	1·908	4·479	21	218	0·148	0·325	12·9	13·8
55-59	17	100	39	456	2·294	4·583	3	33	0·176	0·332	13·0	13·8
(60 and over)	—	15	—	55	—	3·667	—	4	—	0·267	—	13·8
Total	6,788	13,295	16,982	51,139	—	—	1,630	4,202	—	—	—	—

TABLE 11: BROAD DIAGNOSTIC GROUP 10: BRONCHITIS (I.S.C. Nos. 500-502)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	1,694	1,057	0·806	1·170	130	80	0·062	0·089	13·0	13·2
25-29	1,668	1,971	1,815	2,634	1·087	1·335	131	198	0·078	0·100	13·9	13·3
30-34	1,082	2,447	1,180	3,683	1·090	1·506	82	252	0·076	0·103	14·4	14·6
35-39	694	2,611	1,010	3,704	1·454	1·419	50	275	0·072	0·105	20·2	13·5
40-44	625	2,679	913	5,093	1·461	1·900	59	310	0·094	0·116	15·5	16·4
45-49	460	1,898	761	3,652	1·654	1·925	29	200	0·063	0·105	26·2	18·3
50-54	142	670	532	2,358	3·760	3·516	14	105	0·099	0·157	38·0	22·5
55-59	17	100	18	412	1·059	4·141	1	25	0·059	0·251	18·0	16·5
(60 and over)	—	15	—	184	—	12·267	—	5	—	0·333	—	36·8
Total	6,788	13,295	7,923	22,777	—	—	496	1,450	—	—	—	—

*Excludes days beyond first six months of absence.

TABLE 12: BROAD DIAGNOSTIC GROUP 11: OTHER RESPIRATORY DISEASES (I.S.C. Nos. 471-475, 490-493, 510-527, 763, 783)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	3,048	1,762	1·451	1·951	266	147	0·127	0·163	11·5	12·0
25-29	1,668	1,971	2,455	3,531	1·471	1·790	208	289	0·125	0·147	11·8	12·2
30-34	1,082	2,447	1,556	3,506	1·438	1·434	104	294	0·096	0·120	15·0	11·9
35-39	694	2,611	724	4,021	1·043	1·540	53	273	0·076	0·105	13·7	14·7
40-44	625	2,679	1,080	4,186	1·728	1·561	46	243	0·074	0·091	23·5	17·2
45-49	460	1,898	532	3,400	1·157	1·792	34	174	0·074	0·092	15·6	19·5
50-54	142	670	223	1,099	1·576	1·639	14	64	0·099	0·095	15·9	17·2
55-59	17	100	39	401	2·294	4·030	2	12	0·118	0·121	19·5	33·4
(60 and over)	—	15	—	—	—	—	—	—	—	—	—	—
Total	6,788	13,295	9,657	21,906	—	—	727	1,496	—	—	—	—

TABLE 13: BROAD DIAGNOSTIC GROUP 12: DISEASES OF STOMACH AND DUODENUM (I.S.C. Nos. 540-545, 784)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	1,726	1,193	0·822	1·321	122	102	0·058	0·113	14·1	11·7
25-29	1,668	1,971	1,368	2,090	0·819	1·060	85	180	0·051	0·091	16·1	11·6
30-34	1,082	2,447	1,273	2,711	1·176	1·109	76	175	0·070	0·072	16·8	15·5
35-39	694	2,611	396	2,838	0·570	1·087	33	149	0·048	0·057	12·0	19·0
40-44	625	2,679	577	3,077	0·923	1·148	37	164	0·059	0·061	15·6	18·8
45-49	460	1,898	560	2,121	1·217	1·118	33	111	0·072	0·058	17·0	19·1
50-54	142	670	107	580	0·756	0·865	5	27	0·035	0·040	21·4	21·5
55-59	17	100	—	141	—	1·417	—	9	—	0·090	—	15·7
(60 and over)	—	15	—	14	—	0·933	—	2	—	0·133	—	7·0
Total	6,788	13,295	6,007	14,765	—	—	391	919	—	—	—	—

TABLE 14: BROAD DIAGNOSTIC GROUP 13: HERNIA OF ABDOMINAL CAVITY (I.S.C. Nos. 560-561)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	—	15	—	0·017	—	1	—	0·001	—	15·0
25-29	1,668	1,971	—	198	—	0·100	—	6	—	0·003	—	33·0
30-34	1,082	2,447	—	275	—	0·112	—	4	—	0·002	—	68·8
35-39	694	2,611	—	200	—	0·077	—	4	—	0·002	—	50·0
40-44	625	2,679	—	532	—	0·198	—	6	—	0·002	—	88·7
45-49	460	1,898	166	665	361	0·350	1	11	0·002	0·006	166·0	60·5
50-54	142	670	—	—	—	—	—	—	—	—	—	—
55-59	17	100	—	99	—	0·995	—	1	—	0·010	—	99·0
(60 and over)	—	15	—	—	—	—	—	—	—	—	—	—
Total	6,788	13,295	166	1,984	—	—	1	33	—	—	—	—

TABLE 15: BROAD DIAGNOSTIC GROUP 14: OTHER DISEASES OF THE DIGESTIVE SYSTEM
(I.S.C. Nos. 530-539, 550-553, 570-587, 764, 785)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	2,213	1,154	1·053	1·277	149	83	0·071	0·092	14·9	13·9
25-29	1,668	1,971	1,572	2,914	0·942	1·477	113	207	0·068	0·105	13·9	14·1
30-34	1,082	2,447	838	3,139	0·774	1·284	69	218	0·064	0·089	12·1	14·4
35-39	694	2,611	607	3,351	0·874	1·283	36	188	0·052	0·072	16·9	17·8
40-44	625	2,679	389	2,472	0·622	0·922	30	169	0·048	0·063	13·0	14·6
45-49	460	1,898	601	3,329	1·307	1·754	28	146	0·061	0·077	21·5	22·8
50-54	142	670	64	866	0·452	1·291	6	32	0·042	0·048	10·7	27·1
55-59	17	100	—	53	—	0·533	—	4	—	0·040	—	13·3
(60 and over)	—	15	—	—	—	—	—	—	—	—	—	—
Total	6,788	13,295	6,284	17,278	—	—	431	1,047	—	—	—	—

*Excludes days beyond first six months of absence.

TABLE 16: BROAD DIAGNOSTIC GROUP 15: DISEASES OF WOMEN (I.S.C. Nos. 620-689)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	1,073	1,832	0·511	2·028	56	74	0·027	0·082	19·2	24·8
25-29	1,668	1,971	1,008	4,310	0·604	2·185	43	187	0·026	0·095	23·4	23·0
30-34	1,082	2,447	727	4,414	0·672	1·805	27	171	0·025	0·070	26·9	25·8
35-39	694	2,611	933	4,018	1·344	1·539	22	152	0·032	0·058	42·4	26·4
40-44	625	2,679	183	6,003	0·293	2·239	8	134	0·013	0·050	22·9	44·8
45-49	460	1,898	68	3,872	0·148	2·041	6	103	0·013	0·054	11·3	37·6
50-54	142	670	138	1,113	0·975	1·659	3	26	0·021	0·039	46·0	42·8
55-59	17	100	—	24	—	0·241	—	2	—	0·020	—	12·0
(60 and over)	—	15	—	—	—	—	—	—	—	—	—	—
Total	6,788	13,295	4,130	25,586	—	—	165	849	—	—	—	—

TABLE 17: BROAD DIAGNOSTIC GROUP 16: DISEASES OF THE SKIN (I.S.C. Nos. 690-698, 700-716)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	779	736	0·371	0·815	73	37	0·035	0·041	10·7	19·9
25-29	1,668	1,971	565	986	0·338	0·500	53	82	0·032	0·042	10·7	12·0
30-34	1,082	2,447	350	1,217	0·323	0·498	30	90	0·028	0·037	11·7	13·5
35-39	694	2,611	314	1,029	0·452	0·394	17	73	0·024	0·028	18·5	14·1
40-44	625	2,679	280	1,180	0·448	0·440	19	71	0·030	0·026	14·7	16·6
45-49	460	1,898	341	1,035	0·741	0·545	18	67	0·039	0·035	18·9	15·4
50-54	142	670	51	568	0·360	0·847	5	20	0·035	0·030	10·2	28·4
55-59	17	100	—	117	—	1·176	—	4	—	0·040	—	29·3
(60 and over)	—	15	—	50	—	3·333	—	2	—	0·133	—	25·0
Total	6,788	13,295	2,680	6,918	—	—	215	446	—	—	—	—

TABLE 18: BROAD DIAGNOSTIC GROUP 17: DISEASES OF BONES AND ORGANS OF MOVEMENT (I.S.C. Nos. 720-749, 787)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	1,758	1,059	0·837	1·172	111	87	0·053	0·096	15·8	12·2
25-29	1,668	1,971	2,074	2,975	1·242	1·508	123	191	0·072	0·097	17·1	15·6
30-34	1,082	2,447	937	4,378	0·866	1·791	67	243	0·062	0·099	14·0	18·0
35-39	694	2,611	1,407	4,735	2·026	1·814	66	264	0·095	0·101	21·3	17·9
40-44	625	2,679	1,082	7,030	1·731	2·622	66	341	0·106	0·127	16·4	20·6
45-49	460	1,898	1,122	6,341	2·439	3·342	52	289	0·113	0·152	21·6	21·9
50-54	142	670	353	2,404	2·495	3·584	14	104	0·099	0·155	25·2	23·1
55-59	17	100	112	317	6·588	3·186	1	18	0·059	0·181	112·0	17·6
(60 and over)	—	15	—	88	—	5·867	—	2	—	0·133	—	44·0
Total	6,788	13,295	8,845	29,327	—	—	498	1,539	—	—	—	—

TABLE 19: BROAD DIAGNOSTIC GROUP 18: ACCIDENTS ON DUTY (I.S.C. Nos. (N) 800-999)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	1,782	1,048	0·848	1·160	112	61	0·053	0·068	15·9	17·2
25-29	1,668	1,971	1,232	3,005	0·738	1·524	61	127	0·037	0·064	20·2	23·7
30-34	1,082	2,447	904	2,974	0·834	1·216	45	139	0·042	0·057	20·1	21·4
35-39	694	2,611	397	4,894	0·572	1·874	27	182	0·039	0·070	14·7	26·9
40-44	625	2,679	641	4,497	1·026	1·677	30	177	0·048	0·066	21·4	25·4
45-49	460	1,898	1,343	6,326	2·920	3·334	31	166	0·067	0·087	43·3	38·1
50-54	142	670	227	1,596	1·604	2·380	6	50	0·042	0·075	37·8	31
55-59	17	100	8	352	0·471	3·538	1	6	0·059	0·060	8·0	58·7
(60 and over)	—	15	—	—	—	—	—	—	—	—	—	—
Total	6,788	13,295	6,534	24,692	—	—	313	908	—	—	—	—

*Excludes days beyond first six months of absence.

TABLE 20: BROAD DIAGNOSTIC GROUP 19: ACCIDENTS OFF DUTY
(I.S.C. Nos. (N) 800-999)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	1,028	770	0·489	0·852	78	51	0·037	0·056	13·2	15·1
25-29	1,668	1,971	1,258	1,861	0·754	0·944	71	114	0·043	0·058	17·7	16·3
30-34	1,082	2,447	660	2,445	0·610	1·000	43	129	0·040	0·053	15·3	19·0
35-39	694	2,611	439	2,819	0·632	1·080	28	135	0·040	0·052	15·7	20·9
40-44	625	2,679	520	3,263	0·832	1·217	22	149	0·035	0·056	23·6	21·9
45-49	460	1,898	318	3,279	0·691	1·728	17	125	0·037	0·066	18·7	26·2
50-54	142	670	87	1,190	0·615	1·774	4	43	0·028	0·064	21·8	27·7
55-59	17	100	8	141	0·471	1·417	1	6	0·059	0·060	8·0	23·5
(60 and over)	—	15	—	159	—	10·600	—	2	—	0·133	—	79·5
Total	6,788	13,295	4,318	15,927	—	—	264	754	—	—	—	—

TABLE 21: BROAD DIAGNOSTIC GROUP 20: MISCELLANEOUS
(I.S.C. Nos. 000, 240-299, 320-326, 590-617, 750-762, 765-776, 786, 788, 789, 790-795)

Age Group	Exposure to Risk (Man Years)		No. of Days Sickness Absence*		Average Annual Duration Per Person (Days)		No. of Spells		Annual Inception Rate (Spells)		Average Length of Spell (Days)	
	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married	Single	Married
Up to 24	2,100	904	3,830	3,221	1·823	3·566	312	241	0·149	0·267	12·3	13·4
25-29	1,668	1,971	3,012	7,474	1·804	3·789	234	493	0·140	0·250	12·9	15·2
30-34	1,082	2,447	1,470	6,760	1·358	2·765	116	510	0·107	0·209	12·7	13·3
35-39	694	2,611	1,031	7,303	1·485	2·797	65	481	0·094	0·184	15·9	15·2
40-44	625	2,679	825	7,561	1·320	2·820	66	439	0·106	0·164	12·5	17·2
45-49	460	1,898	798	4,232	1·735	2·230	51	292	0·111	0·154	15·6	14·5
50-54	142	670	420	1,842	2·968	2·746	12	103	0·085	0·154	35·0	17·9
55-59	17	100	15	238	0·882	2·392	1	19	0·059	0·191	15·0	12·6
(60 and over)	—	15	—	15	—	1·000	—	1	—	0·067	—	15·0
Total	6,788	13,295	11,401	38,646	—	—	857	2,579	—	—	—	—

*Excludes days beyond first six months of absence.

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OLFACTION TESTS

BY

I. DINGWALL FORDYCE

From the Nuffield Department of Occupational Health, University of Manchester

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A simple olfaction test, based on graded solutions of phenol in liquid paraffin, was examined for repeatability. Of 98 persons tested, 79 repeated their initial result to within ± one grade on a second occasion. The time interval between tests (varying from a few hours to three weeks), observers, age, smoking habits, or initial grade did not appear to affect the degree of repeatability. There was a tendency to improve the grading on the second occasion.

Tests on 147 persons provided no evidence of an association between cigarette smoking and acuity of smell. There was an indication that pipe smoking had an adverse effect. An association between age and acuity was shown.

The measurement of anosmia in field studies presents considerable difficulty. No accurate and simple tests suitable for such studies have been perfected; all are liable to subjective bias and little is known about individual variation from time to time.

An attempt has been made to examine this problem more closely, especially in regard to individual repeatability, using one very simple test. At the same time a guide has been sought to the possible correlation of acuity of smell with age and smoking habits.

Method

The method was kept as simple as possible with a view to use in group studies of men at their places of work.

The olfacts were solutions of phenol in liquid paraffin, sp. gr. 0·880 (Proetz, 1941). The concentrations used are shown in Table 1; eight grades of olfact were used.

TABLE I

CONCENTRATIONS OF SOLUTIONS OF PHENOL IN LIQUID PARAFFIN

Relative Strength of Olfact	Concentration (g./l.)	Grade of Olfact
1	0·0192	1
2	0·0384	2
5	0·0960	3
10	0·1921	4
25	0·4802	5
50	0·9605	6
100	1·9210	7
200	3·8420	8

These solutions had a characteristic odour, were not irritating to the mucous membrane, were reasonably stable, and were of low volatility. They were kept in wide-mouthed, glass-stoppered bottles, filled almost to the stopper. Tests were begun with the weakest solution and continued in order until smell was noticed. The room used was not especially free of adventitious odours; it was an office situated near laboratories. The subjects were not medically examined.

The test population included porters, cleaning women, technicians, secretaries, and medical staff. Everyone who was asked to do so took part. The repeat tests were randomly selected.

The first 100 persons, allocated at random to two observers, returned for a second test at intervals ranging from within 24 hours to 12 days. Three weeks later 35, selected at random, were tested a third time. An additional 49 persons were tested once.

Results

Two of the series of 100 duplicate tests were discarded. One subject had developed an acute nasopharyngitis between tests. The other gave wildly oscillating answers, claiming at one time to notice a smell at two olfacts but none at 200.

The results of the tests on the remaining 98 persons, 35 of whom were later tested for a third time, were examined for repeatability.

Repeatability.—Twenty-eight people gave the same grade of olfact at both tests, 51 differed by

TABLE 2
REPEATABILITY AND TIME INTERVAL

	Time Interval	Difference in Grade of Olfact				No. in Group
		0	±1	±2	±3 and More	
98 duplicate tests						
	Within 24 hours	11	27	6	2	46
	2-4 days	17	21	7	2	47
	6-12 days	0	3	0	2	5
35 triplicate tests						
First and second test	1-4 days	14	12	7	2	35
Second and third test	3 weeks	14	15	4	2	35

± one grade, 13 by ± two grades and six by ± three or more grades.

It was thought possible that the time interval between tests, experience, observers, age, smoking habits, or initial grade might affect the degree of repeatability.

(*a*) *Time Interval.*—The second tests were made at intervals ranging from within 24 hours to 12 days and the third test three weeks later. The length of time which elapsed between tests was found to have little effect upon the consistency of the results (Table 2).

(*b*) *Experience.*—There was a tendency to record a higher degree of acuity at the second test than at the first. Forty-eight persons improved their rating, 22 deteriorated, a difference which is probably significant ($p<0.01$, $\chi^2=9.7$). This bias towards improvement rather than deterioration was also apparent in the third test; details are given in Table 3.

TABLE 3
POSSIBLE EFFECT OF EXPERIENCE

Apparent Degree of Acuity	Duplicate Tests (98)	Triplicate Tests (35)	
		First and Second	Second and Third
Constant	28	14	14
Improved	48	12	12
Deteriorated	22	9	9

(*c*) *Observer Differences.*—The degree of consistency was found to be similar whether both tests were carried out by the same or different observers. Nor was there any evidence of difference between observers as measured by improvement or deterioration at the second test. Details are given in Table 4.

TABLE 4
DIFFERENCE BETWEEN OBSERVERS

Consistency	Same Observer Both Tests (31)	Different Observer at Second Test (67)	Different Observers	
			A First B Second (40)	B First A Second (27)
Constant	9	19	9	10
Improved	17	31	19	12
Deteriorated	5	17	12	5

Smoking and Acuity of Smell.*—In all 147 persons were tested, of whom 90 did not smoke, 46 smoked cigarettes only, 10 smoked a pipe, and one smoked only cigars.

In order to examine the possibility that smoking had a deleterious effect upon the acuity of smell the mean grades of olfacts of non-smokers, cigarette smokers, and others were compared (Table 5). There was no evidence of any difference between cigarette smokers and non-smokers. There was, however, an indication of a possible decrease in acuity among other smokers, but the numbers involved were too small for firm conclusions.

Age and Acuity of Smell.—For the purpose of investigating the effects of ageing the 10 pipe smokers, the cigar smoker, and one woman aged 37 with virtual anosmia were excluded from the sample.

Details of the mean olfacts in age groups of the remaining 135 persons are given in Table 6.

Although an overall decrease in acuity with increasing age is apparent it is difficult to explain, other than by chance, the very low grading in the age group 35-44. However, the sharp decline in this age group was apparent in all sub-divisions of the total sample.

Conclusions and Summary

The olfaction test, described above, although crude and subjective proved repeatable to within ± one grade by 81% of the sample of 98 persons. No appreciable difference in accuracy was noticed whether the time interval was less than 24 hours or as long as three weeks. When there was a change,

*The age distribution of the subjects was not representative of the general population and this may, in part, account for the difference found in smoking habits. Among the younger men, who were over-represented in the sample, about 50% were smokers, whilst among those over 45 the incidence rose to about 65%. Of the women over the age of 25 approximately 38% smoked.

TABLE 5

MEAN GRADES OF OLFACTS OF SMOKERS AND NON-SMOKERS

Smoking Habits	Age Group				
	15-24	25-34	35-44	45-54	55-70
Number of non-smokers*	31	18	16	16	8
Mean olfact	2·6	2·6	7·9	4·6	5·1
Number of cigarette smokers	11	11	9	9	6
Mean olfact	3·1	2·7	9·3	2·2	4·3
Number of other smokers	1	1	4	3	2
Mean olfact	2	5	21·1	7·1	10 and 100

*One woman of 37, graded · 200, has been excluded.

TABLE 6

MEAN GRADES OF OLFACT IN THREE AGE GROUPS

Group		Age Group		
		15-34	35-44	45-70
Total* (excluding pipe smokers, etc.)	Mean olfact	2·7	8·4	4·1
	Variance	4·9	136·3	11·5
	No.	71	25	39
First Series	Mean olfact	3·0	8·0	4·2
	No.	54	12	28
Second series	Mean olfact	1·7	8·8	3·9
	No.	17	13	11
Men	Mean olfact	2·8	10·7	4·2
	No.	27	11	12
Women	Mean olfact	2·6	6·6	4·2
	No.	44	14	27

*Difference in means at ages 15-34 and 45-70 is probably significant ($p > 0{\cdot}01$, $t = 2{\cdot}62$).
Difference in means at ages 35-44 and 45-70 is possibly significant ($p > 0{\cdot}05 > 0{\cdot}01$, $t = 2{\cdot}1$).

the tendency was for the grade to improve, perhaps as a result of previous knowledge of the particular smell. There was no evidence that individual observers influenced the results. Age, smoking habits, and initial degree of acuity had no measurable effect upon the consistency of results found in this sample.

There was no evidence of any correlation between cigarette smoking and acuity of smell. Pipe smoking, however, may possibly have a deleterious effect although the number of such smokers in this sample was too small for any valid conclusions to be drawn.

Although there was a significant decrease in acuity of smell with increasing age the results were not straightforward. The age group 35-44 was more affected than the older groups, an effect which was noticed in all sub-sections of the sample. There is no ready explanation for this possibly chance phenomenon.

This test appears to be of practical use in field studies of moderately large groups of men. The negative correlation between acuity of smell and age (slight but probably conclusive) would have to be taken into account in analysis of results and choice of controls. Until further evidence is available it would be wise to assume that there is also a negative correlation between acuity of smell and pipe smoking although cigarette smokers appear to be unaffected.

REFERENCE

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ANOSMIA IN ALKALINE BATTERY WORKERS

BY

R. G. ADAMS and NORMAN CRABTREE

From Alkaline Battery Company Limited, Redditch, and The United Birmingham Hospitals

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The sense of smell of 106 alkaline battery workmen exposed at their work to cadmium and nickel dust has been compared with a control group of 84 men matched for age. The battery workers reported significantly more anosmia than the controls (15% to zero) and did less well in the phenol smelling test (27·3% to 4·8%). Cadmium proteinuria was found in 17 of the battery workers, 11 of whom showed virtual anosmia. Figures of recent concentrations of cadmium and nickel in the atmosphere are given. The noses of 85 battery workers and 75 controls were examined. Signs of non-specific chronic irritation were more frequent in the battery workers but no significant relationship was established between this appearance and the presence of anosmia. It is concluded that the anosmia is due to exposure to cadmium or nickel dust or a mixture of the two.

This is a study of olfactory acuity in a group of men employed in alkaline battery manufacture, and is divided into two parts. Part I reports on the incidence of anosmia, and Part II deals with the results of macroscopic examination of the men's noses. This was undertaken to determine whether specific changes could be observed in the nasal mucosa and whether these, if present, could be related to olfactory acuity.

Two powders are used in the manufacture of alkaline batteries: cadmium powder, which contains 60-64% of cadmium oxide and 16-18% of ferrous iron; and nickel powder, which contains 72% of nickel hydroxide and 18-20% of graphite.

Anosmia was first noted in this factory in 1948. In a survey at this time it was found that of 68 men examined, 13 (19%) had anosmia and 18 (26%) had proteinuria (C. L. Potts, personal communication). Shortly before this Friberg (1948) reported on his findings of proteinuria and emphysema in alkaline battery workers in Sweden; and he also noted that some of these men had anosmia. In his paper on emphysema and proteinuria in men casting copper-cadmium alloys, Bonnell (1955) noted that some of the men complained of nasal soreness, but none of them appeared to have lost the sense of smell.

As a result of these discoveries, the men exposed to cadmium in this factory were offered annual medical examinations. During these examinations, which were started in 1956, particular attention was paid to renal, respiratory, and olfactory function.

PART I

The results in this part of the paper are taken from the records of the examinations carried out by one of us (R.G.A.) in 1958-1959. One hundred and six men were examined, six individuals being seen each week.

A control group was obtained in 1958 from a neighbouring engineering factory in which there was no specific hazard. The names of 100 men in this factory, in approximately the same distribution in five-year age groups as the battery workers, were randomly selected. Out of this group of 100, 84 attended for examination.

Olfactory Acuity

In the absence of an objective test, the degree of olfactory acuity was measured by two methods.

(1) Each man was asked "What is your sense of smell like?", and the answer recorded as Good, Diminished, or None.

(2) A modification of the Proetz (1941) olfactometer. Phenol in liquid paraffin was the only substance used, the strength of different bottles being measured in olfacts. One olfact is defined as the minimum concentration of the substance in solution, which can be perceived by a large number of normal individuals, and the concentrations used were 1, 2, 5, 10, 25, 50, 100, and 200 olfacts of phenol. A man's olfactory acuity to phenol was recorded as the lowest concentration that he could detect. If he could not smell 200 olfacts his acuity was recorded

TABLE 1a
BATTERY WORKERS

		Olfactory Acuity (in Olfacts of Phenol)									Totals
		1	2	5	10	25	50	100	200	200+	
Symptomatic grades of smelling	Good	15	18	13	6	7	3	2	1	2	67
	Diminished	0	0	2	2	4	1	1	2	11	23
	None	0	0	0	0	0	0	0	0	16	16
Totals		15	18	15	8	11	4	3	3	29	106

TABLE 1b
CONTROL WORKERS

		Olfactory Acuity (in Olfacts of Phenol)									Totals
		1	2	5	10	25	50	100	200	200+	
Symptomatic grades of smelling	Good	17	17	18	7	4	2	1	0	1	67
	Diminished	2	1	3	3	1	2	1	1	3	17
	None	0	0	0	0	0	0	0	0	0	0
Totals		19	18	21	10	5	4	2	1	4	84

as 200+, and this was accepted as virtual anosmia. (No precautions were taken to ensure an odourless room, smoking was not prohibited before the test, crusts in the nose were not removed, and the nose was not treated with ephedrine in the presence of obstruction. The object of these modifications was to simplify the test for purposes of field study.)

Results

Table 1a shows how the battery workers in the three symptomatic grades responded to the smelling test.

The Table shows that there were 16 battery workers (15%) who claimed to have no smell sense, all of whom had an acuity to phenol of 200+. In all, there were 29 battery workers with acuity to phenol of 200+ (27·3%).

Of the 23 men who reported diminished smell sense, 11 had an acuity to phenol of 200+ (*i.e.* virtual anosmia) suggesting that nearly half this group may have anosmia without realizing it. The remaining 12 responded to the phenol test in widely differing degrees, thus emphasizing the difficulty of assessing hyposmia quantitatively.

Table 1b shows how the control workers in the three symptomatic grades responded to the smelling test.

From this Table it can be seen that none of the control workers claimed to have lost their sense of smell, as compared with 16 in the battery group. Seventeen of the control workers reported diminished smell sense, but only three of these had a phenol acuity of 200+. In all there were four control workers with an acuity to phenol of 200+ (4·8%).

When the results in the two groups are compared, it is apparent that the alkaline battery workers reported significantly more anosmia than the control workers (15% compared with 0%), and responded less well to the phenol test (27·3% with phenol acuity of 200+ compared with 4·8%).

Of the many factors which may affect the sense of smell, it was thought that smoking and age might be important, and these two factors were therefore studied. It was found that a greater proportion of the battery workers smoked, especially in the younger age groups. For statistical purposes, therefore, the most satisfactory comparison is made between smokers aged 35 and over.

TABLE 2
PERCENTAGE AGE DISTRIBUTION, SMOKERS AGED 35 AND OVER, AND MEAN NUMBER OF CIGARETTES PER SMOKER PER DAY

Age (Years)	Controls			Battery Workers		
	No.	Per cent.	Mean No. of Cigarettes	No.	Per cent.	Mean No. of Cigarettes
35 to 44	18	35	17·2	22	36	13·7
45 to 54	18	35	20·8	25	41	16·9
55 and over	15	29	18·3	14	23	14·8
35 and over	51	100	18·8	61	100	15·2

Table 2 shows that the controls smoked more than the battery workers. If any bias were introduced by these differences, it would tend to operate against finding a higher incidence of anosmia among the battery workers.

TABLE 3

ACUITY OF SMELL TO PHENOL AMONG SMOKERS AGED 35 AND OVER

Grades of Acuity in Olfacts	Controls	Battery Workers
1 to 5	33	19
10 to 200	15	20
200 +	3	22
Totals	51	61

($p < 0.001$, $\chi^2 = 16.9$)

The figures in Table 3 confirm the previous finding of more anosmia among the battery workers. The greater incidence, which is highly significant, cannot be attributed to differences in age or smoking habits.

Anosmia and Proteinuria.—The association between proteinuria and exposure to cadmium is established (Friberg, 1950; Smith, Kench, and Lane, 1955; Bonnell, 1955; Smith and Kench, 1957).

The properties of the protein in the urine of some of the workers in the factory in this study, have recently been investigated in great detail by Smith, Wells, and Kench (1961), and these authors confirm and add to the findings of Friberg and Bonnell.

Out of the 106 battery workers seen in 1958-1959, 17* were found to have proteinuria (as shown by urine testing with 3% salicyl-sulphonic acid).

Exposure

In the process of alkaline battery manufacture it is important that the powders used should be very dry, and as a consequence the process is a dusty one. Contamination of the atmosphere is controlled by exhaust ventilation, and in this factory, extensive installations have been developed over the years.

Regular cadmium-in-air estimations have been carried out at fixed sites in the factory since 1957, and the results for the years 1957 and 1960 are shown below. These give an indication of the sort of conditions prevailing during the past four years. (Before 1957 the air sampling had not been carried out at exactly the same sites and the readings are not therefore comparable.)

Also shown below are the results of recent nickel-in-air estimations from some similar sites, at which the nickel powder was worked.

Cadmium-in-air (mg. Cd/10 cu.m.)		Nickel-in-air (mg. Ni/10 cu.m.)
1957	1960	1960
27·6	4·6	—
1·60	4·10	—
0·65	1·18	0·016
16·90	0·51	0·024
0·45	0·78	0·16
0·28	0·34	0·016
3·70	6·65	—
1·40	1·18	—
3·80	1·82	—
2·20	1·87	0·046
5·50	1·44	0·56
3·60	4·10	—
4·30	0·41	—
5·70	0·74	—
4·30	1·24	—
—	0·65	0·042

TABLE 4

ANOSMIA RELATED TO PRESENCE AND ABSENCE OF PROTEINURIA AMONG BATTERY WORKERS

Clinical Group	No. of Men in Group	Virtual Anosmia to Phenol		Symptomatic Grading Diminished or None	
		No.	Per cent.	No.	Per cent.
Proteinuria present	17	11	65	12	70
Proteinuria absent	89	18	20	27	30

Of the 17 men with proteinuria, there were 11 who showed virtual anosmia to phenol, and 12 who claimed diminished or complete loss of smell. Of the 89 men without proteinuria, the corresponding figures were 18 (20%) and 27 (30%), and when compared with the previous figures, indicated a significant positive association between the incidence of proteinuria and anosmia (Table 4).

The cadmium readings tend to be variable but the overall impression is that of a reduction in the amount of cadmium dust in 1960 compared with 1957. Both sets of cadmium readings are, however, considerably higher than those for nickel.

The length of exposure of the workers in this study varied from a few weeks to over 30 years, and 28 of the workers had been employed in a similar factory which closed down 13 years ago. Although no atmospheric monitoring was carried out in this factory, conditions were known to have been much dustier than in the present one. It is worth noting

*One of these men, who was known to have been excreting protein for at least 10 years, was found to have normal renal function, after extensive investigations in the Department of Experimental Pathology, Queen Elizabeth Hospital, Birmingham.

that 13 of these 28 workers had a phenol acuity of 200, compared with 16 of the remaining 78. The fact that these 28 men tended to be older than the rest of the group may have had some effect, but it is unlikely alone to account for the large difference. In addition 13 of the 17 men shown to have proteinuria, had worked in the factory which closed down. It does seem likely therefore, that many of the abnormalities found in this study were due to conditions unlike those obtaining today. These facts and suggestions are of no help in attempting to discover which of the ingredients of the two powders is responsible for producing anosmia, and no answer to this problem has been obtained.

PART II

Changes in the Nasal Mucosa

Ulceration of the nasal mucosa accompanied by burning sensations in a group of cadmium-platers was described by Mancioli (1940). The nasal passages were examined by one of us (N.L.C.) to see whether such changes existed in our series, whether they could be due to some specific action of powders to which alkaline battery workers are exposed, and related to the tendency to lose the sense of smell.

As an examination of this kind depends on a description of visible changes it was considered important that they be recorded by one individual examiner within a short space of time, and that at the time of the examination the examiner should not be unconsciously biased by knowing the identity of each man. Eighty-five workers and 75 control subjects were therefore presented at random in two sessions a few days apart at the end of 1958.

Findings at Examination.—As with any sample of population there were variations in anatomy and signs of infection or allergy in varying degrees. Those which were clearly and recognizably due to non-specific causes were recorded but excluded from analysis.

In many cases, signs of irritation from inhaled material were found and recorded. Greyish dust was found on the mucosa in some cases where underlying pathology was not detected. In all cases the areas affected corresponded to the point at which the jet of inspired air impinged on the membrane, although this situation varied according to the detailed shape of the nasal passages. Where changes were seen the septum was involved in an oblique line corresponding to the air stream, the principal other sites being the anterior ends of the middle and inferior turbinate bones.

In mild cases there was a small localized area of whitening with slight thickening of the mucosa, indicating fibrosis beneath. The size varied from 0·5 to 3 cm. In more advanced cases the centre was slightly depressed with shallow ulceration leading occasionally to the formation of dry crusting. In one case of prolonged exposure, very extensive dry crusting involving nearly all the nasal mucosa was seen. This was the only case in which any abnormality of the olfactory area in the upper one-third of the nasal cavity was detected, and here it was an extension of an even more marked atrophy and crusting of the respiratory portion of the mucosa. In no other case did the changes extend onto the olfactory area.

As an extension of this examination, and at another time, an area of affected mucosa was removed from a known battery worker. Microscopic examination by Dr. Brewer of the Pathological Department, University of Birmingham, showed a mild non-specific chronic inflammation of the submucosa with a few small loose focal accumulations of lymphocytes and a few widely scattered eosinophils.

TABLE 5

Workers	Group			
	A	B	C	Total
Battery	36	43	5	84
Control	45	29	1	75

Analysis of Clinical Findings.—According to the changes in the mucosa, cases were grouped into three categories.

A. No changes which could be ascribed to inhalation of irritant material.

B. Definite signs of irritation of a mild degree.

C. Marked changes unquestionably due to the inhalation of an irritant material producing definite destructive change.

Table 5 shows the distribution of battery and control workers in these three groups.

It is clear from the presence of control workers in the B and C Groups that the changes noted cannot be ascribed to irritation specifically from cadmium and nickel dusts. It is worth observing that there are more of the battery workers in Groups B and C than in Group A, but more of the control workers in Group A than in B and C, *i.e.* irritative changes in the nose are more common amongst the battery workers.

The relation of the phenol acuity in the alkaline battery workers to the presence of physical changes in the nose is shown in Table 6, the columns A, B, and C being the clinical groups previously described (five of the 84 individuals had not been tested for phenol acuity).

TABLE 6

Phenol Acuity	A	B	C
1 to 25	23	20	1
50 to 200	1	6	0
200 +	10	14	4

Analysis (by attaching an approximate logarithmic scale to the degrees of phenol acuity, where 1 corresponds to 0 and 200 + corresponds to 8) shows that the mean olfactory sensitivity in the A Group is 3·62 and in the combined B and C Groups is 4·54 and is of no significance.

We can conclude that the presence of the changes described in the nasal mucosa are of no value in assessing the degree of hyposmia.

The relation of symptoms of diminished olfactory acuity to the physical changes, shown in Table 7 also show no significance (four of the 84 individuals had not been previously questioned on their sense of smell).

TABLE 7

Olfactory Acuity	A	B	C
Good	20	25	2
Diminished	7	9	1
None	7	7	2

DISCUSSION

In the factory under examination it has been fairly common knowledge that "the powder irritates the nose", but in fact only two individuals have complained of or mentioned a sore nose during the course of medical examination; this in spite of the fact that attention has been directed towards the nose by questions about the sense of smell. This suggests that any such symptoms have been considered too trivial to mention.

To establish the existence of anosmia, or the degree of hyposmia is another matter, for there is no completely satisfactory and objective test of olfaction. We are dependent on a subjective test requiring the willing and unbiased co-operation of the individual. The phenol test used in this investigation appears to be a useful and repeatable clinical guide. It would be invalid under conditions where the individual might be tempted, consciously or unconsciously, to mislead. The corollary is equally true, that it would be impossible to prove the existence of the disability by such a subjective test.

Apart from the consistency and standardization of the strength of the odour, it is vital that the odour used should be detectable by olfaction alone, and not by taste or by irritation of the trigeminal nerve. We are accustomed by habit and convention to synthesize these three related sensations. The "taste" of food is a combination of taste and olfaction, and for the identification of ammonia the irritation of the trigeminal nerve is at least as important as, and certainly more dramatic than olfaction.

The phenol test has no value as a quantitative assessment of the degree of hyposmia. Most of the men with a good sense of smell were able to detect phenol in very high dilution, while many of those with diminished smell were unable to detect it even in the highest concentrations. It is interesting that the failure to detect phenol even in high concentration occurred in several men who were not aware of any significant disability.

Our findings show that exposure to alkaline battery dust can seriously impair the sense of smell, and also that there is a tendency towards changes of a chronic irritative nature in the nasal mucosa. It is quite clear, however, that these changes, while more common in the battery workers, are not specific, and their nature and degree are of no value in indicating the presence of anosmia.

Very few cases, even those with prolonged exposure, showed changes of a degree likely to cause any other disability. Irritation did not form a subject for complaint. In Mancioli's cases irritation, cough, dryness of the pharynx, bronchitis, and broncho-pneumonia, as well as gastrointestinal disturbances, were described. In only a few of the cases in our series, did the appearances in the nose correspond to those described by Mancioli.

The circumstances of their employment make it impossible to identify the particular ingredient in the dust that is responsible for the anosmia in the workers in this study. Iron oxide and graphite may be safely excluded since they are frequently encountered in industry, and anosmia has not been reported.

The reduction of the sense of smell in a large number of workers engaged on the production of nickel by an electrolytic process has recently been reported in Russia (Tatarskaya, 1960). This has not been reported elsewhere and as far as we have been able to determine in our inquiries, it has not been experienced in this country.

The positive correlation of anosmia with proteinuria in our series suggests that cadmium oxide is the offending dust, but it may well be that any irritant substance which is breathed for long periods either as a fine dust or aerosol can impair the sense of smell.

It is interesting to note that inhalation of cadmium fume affects the lower respiratory tract (Bonnell, 1955). The size of the particulate matter which is inhaled may be a factor influencing the site of the lesion, the larger particles of powder being held by

the mucous blanket covering the nasal mucosa while the more finely particulate fume more readily reaches and affects the lungs.

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THE KIDNEY IN LEAD POISONING

BY

ZDENKO RADOŠEVIĆ*, MARKO ŠARIĆ†, TIHOMIL BERITIĆ‡, and JELICA KNEŽEVIĆ§

From the Institute for Medical Research, Yugoslav Academy of Sciences and Arts, Zagreb

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Kidney damage due to lead is still an interesting problem of industrial toxicology. In spite of abundant literature data, much still remains to be explained. There are controversial opinions, not only on the type of renal lesions due to lead, but also on whether lead affects the kidney at all.

In this paper our clinical observations on the effect of lead upon the kidney in 53 patients suffering from lead poisoning are presented. In 44 patients (40 men and four women) lead poisoning was due to occupation, and in nine (five men and four women) to the use of lead-glazed pottery. The length of exposure varied from two months to 35 years. In all cases the diagnosis of lead poisoning was made clinically and confirmed by laboratory tests.

Permanent changes in the form of chronic nephropathy were observed in only two patients. These were the two cases in which exposure to lead was the longest and most intense. Twenty-three patients showed functional renal lesions tending to normalize. In addition to the cases of organic nephropathy, blood pressure was persistently raised in one further patient; in two patients a raised blood pressure was observed only in the acute stage of poisoning.

On the basis of these findings we consider that lead intoxication can cause renal lesions. These lesions are for the most part functional and temporary. In cases of long and severe exposure and repeated lead intoxication, organic renal lesions seem possible. The disturbances of renal function observed in this study may be ascribed to disordered intrarenal circulation, due to the spastic effect of lead on intrarenal blood vessels, and to a direct toxic or indirect hypoxic effect of lead on the tubules.

When investigating renal function, we have observed that the timing of individual tests is of paramount importance. Some lesions are subject to changes in the natural course of lead poisoning, and unless this is borne in mind, apparently contradictory results may be obtained.

The question as to whether lead produces kidney damage has been discussed for nearly 100 years, and never answered satisfactorily. The problem is still important, both from theoretical and practical points of view, and in Yugoslavia in particular, where not only industrial workers but also peasants are exposed to lead comparatively often.

Published data on the effect of lead upon the kidney are often contradictory. The earlier writers were convinced that lead caused kidney damage and did not hesitate to use a special nosologic term "lead kidney" (Bleiniere, *nephropathia saturnina*) as if it were an accepted fact (Lancereaux, 1881; Leyden, 1883; Gayler, 1887; Oliver, 1914; Volhard and Fahr, 1914). More recently authors, especially in America, are doubtful about the existence of such damage (Belknap, 1936; Mayers, 1947; Humperdinck, 1948; Johnstone, 1948; Greenfield and Gray, 1950).

The approach to the problem may have accounted for such divergent opinions: on the one hand, the lack of definite diagnostic criteria and reliable statistics on the frequency of the so-called lead nephropathy but an experience obtained by observations of a great number of persons with a long and severe exposure to lead and suffering

* Associate Professor of Medicine, Medical Faculty, University of Zagreb, Zagreb.

† Research Associate, Institute for Medical Research (incorporating the Institute of Industrial Hygiene), Yugoslav Academy of Sciences and Arts, Zagreb.

‡ Senior Research Associate, Recognized Teacher of Industrial Hygiene, Institute for Medical Research (incorporating the Institute of Industrial Hygiene), Yugoslav Academy of Sciences and Arts, Zagreb.

§ Assistant, Institute for Medical Research (incorporating the Institute of Industrial Hygiene), Yugoslav Academy of Sciences and Arts, Zagreb.

from severe lead poisoning; on the other hand, more reliable modern diagnostic procedures based on well-developed renal function tests but little experience concerning long exposure to lead and severe lead poisoning.

There are modern writers who do consider kidney damage due to lead to be possible. Cantarow and Trumper (1944) drew attention to difficulties in the interpretation of results, and pitfalls in the assessment of the findings, especially those based on insufficiently checked clinical observations and post-mortem findings. Nevertheless, they considered that the opinion of the medical authorities who recorded manifest renal lesions due to lead should be taken into consideration. Lane (1949) attempted to explain the contradictory statements on this problem by studying workers' health and working conditions in Britain in the course of the industrial development of that country. In his opinion, the changing intensity of exposure to lead in the course of time may have accounted for great differences between former and present-day findings.

Bell, Williams, and Cunningham (1925) reinforced the belief that lead could produce renal lesions. For therapeutic purposes they administered colloidal lead solution intravenously to patients suffering from malignant tumours. Pronounced lesions of the renal tubular epithelium were observed. However, from the work of these authors, nothing can be concluded about the kidney changes which may occur in the course of a prolonged exposure to lead.

In connexion with the role of lead in the aetiology of kidney lesions special attention should be drawn to the investigations of Australian workers. As early as the first decade of this century a higher incidence of chronic nephritis was observed in Queensland, Australia than in other parts of the country. From the first observation it was believed that the disease was a late result of excessive lead absorption in childhood. The investigators dealing with this problem (Nye, 1933; Cilento, 1932; Murray, 1939) came to the conclusion that at least some of these cases of chronic nephritis could be attributed to the effects of lead. There was evidence of past lead poisoning in about a third of the cases examined. In the remaining cases, it was believed that sub-clinical lead poisoning might be responsible for the incidence of chronic nephritis, but there was no direct evidence to prove it. In later publications dealing with the same problem (Henderson, 1954; Henderson, 1955; Henderson and Inglis, 1957) the causal link between lead poisoning in childhood and chronic nephritis was again considered. In a comprehensive review, Henderson (1958) supported his opinion of the lead aetiology of Queensland chronic nephritis by the finding of a high percentage of renal disease in persons known to have had lead poisoning in childhood. There was a comparatively frequent history of childhood saturnism in the chronic nephritis group investigated, and bone lead content was raised significantly in the fatal cases of the same group. Henderson laid great emphasis on the fact that these findings occurred only in a group of cases which had been separated on the basis of clinico-pathological findings different from those common in renal disease of generally accepted aetiology, such as glomerulonephritis and pyelonephritis.

There is also considerable difference of opinion on the mechanism of the noxious effect of lead on the kidney and the pathogenesis of renal lesions. Both experiments on animals and clinical and histopathological studies on man have revealed different pathological changes, such as vasculopathy (Hoffa, 1883; Lancereaux, 1881; Leyden, 1883; Gayler, 1887; Volhard and Fahr, 1914; Brogsitter and Wodarz, 1922; Battaglia, 1927; Aiello, 1931; Wirtschafter, 1933; Micheli, 1935; Izar, 1941; Vigliani, 1950), primary tubular lesions (Jores, 1902; Flury, 1934; Beintker, 1929), glomerulitis (Coen and D'Ajutolo, 1888), and interstitial nephritis (Oliver, 1914).

It was hoped that the introduction of the clearance methods into the functional diagnosis of renal disease would throw more light on kidney trouble due to lead. However, the results obtained were again divergent. Crepet, Chiesura, and Gobbato (1953) concluded that renal lesions due to lead manifested themselves either in changes of the filtration membrane or in decreased intrarenal blood circulation brought about by a spasm of afferent arterioles. Radulescu, Dinischiotu, Maugsch, Ionescu, and Teodorescu-Exarcu (1957) observed a diminished filtration rate during attacks of lead colic, which they ascribed, at least in part, to interstitial oedema and diminished intrarenal blood circulation. In severe poisoning they assumed that interstitial nephritis was produced, as a result of increased permeability of blood vessels.

A number of Italian writers (Aiello, 1931; Micheli, 1935; Izar, 1941; Vigliani, 1950) favour the idea that lead has a primary vascular effect. Repeated angiospasm can in the end produce organic lesions, from fibrinoid necrosis of the arteries to the contracted nephrosclerotic kidney (Rizzo and Sbertoli, 1956; Baldi and Sbertoli, 1957).

From these data it is concluded that modern writers, using up-to-date methods for the study of the kidney are also inclined to think, however different their results are, that renal lesions due to

TABLE 1

AGE, SEX, AND DURATION OF EXPOSURE IN 53 CASES OF LEAD POISONING

Age	Duration of Exposure to Lead (years)																			
	< 1		1–2		3–4		5–9		10–14		15–19		20–24		25–29		30–35		Unknown	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
20 —	5	–	4	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
25 —	4	–	4	–	5	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
30 —	1	1	2	–	1	1	1	–	–	1	–	–	–	–	–	–	–	–	1	1
35 —	1	–	1	–	1	1	1	–	1	–	–	–	–	–	–	–	–	–	–	–
40 —	–	–	1	–	–	–	1	–	1	–	–	–	–	–	–	–	–	–	1	1
45 —	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	1	–
50 —	–	–	–	–	–	–	1	–	–	–	–	–	1	–	–	–	–	–	2	–
55–60	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–
33·9 (average)	11	1	13	–	7	2	5	–	2	1	–	–	1	–	–	–	1	–	5	4

lead are possible. This has also been proved by biochemical studies (Dolowitz, Fazekas, and Himwich, 1937) and the study of enzyme dysfunction (Goldblatt and Goldblatt, 1956) and selective aminoaciduria (Clarkson and Kench, 1956; Wilson, Thomson, and Dent, 1953; Granati and Scavo, 1957) appearing in the course of lead poisoning.

We undertook the investigations now described in order to obtain more evidence about the effect of lead upon the kidney. We also wished to explain the cause of such great differences of opinion about the mechanism of the action of lead on the kidney.

Material and Methods

In the course of the past five years we have had under observation 53 patients suffering from lead poisoning. Their age, sex, and duration of exposure are given in Table 1.

Forty-four patients (40 men and four women) suffered from occupational lead poisoning (manufacture of electric accumulators and white lead, and the lead-glazing of earthenware). Lead intoxication in nine patients (five men and four women) was due to the use of lead-glazed pottery for household purposes. Two patients with occupational saturnism (lead-glazing) also used lead-glazed pottery in their households, thus combining two sources of poisoning.

Lead-glazed pottery as a source of lead poisoning in Yugoslavia has been described by Beritić and Djurić (1956). It is well known that such pottery is used in the villages for such purposes as milk fermentation, pickling of vegetables, and wine and brandy preparation.

The period of occupational exposure to lead in the patients observed varied from two months to 35 years. Only two patients had a longer exposure than 14 years (20 and 35 years respectively).

In the cases of non-occupational lead poisoning (due to the household use of lead-glazed pottery), it was impossible to obtain an accurate estimate of the duration of exposure; the contamination was generally long and of varied intensity dep ding upon such factors as the season and the *p*H of the pottery contents.

Lead poisoning was divided into the types described by Johnstone (1948).

Stippled cells were determined by the Hamel method (1900) in all patients.

Reticulocytes were also determined in all patients. A modified Wolfer method was used (Schudel, 1938).

Lead in blood analyses were made in 44 patients by the modified dithizone method (Weber, Voloder, and Vouk, 1952).

Urinary porphyrins were determined by the modified Fisher method (Weber and Ruždić, 1951); this method was used in 44 patients.

All these analyses were made immediately after the admission of each patient to hospital.

Urine analyses were made of all patients on the first or second day of admission.

The urine concentration test was carried out in 21 patients by the modified Fishberg method (Fishberg, 1939) as follows: the evening meal contained no liquid; no fluid was consumed from 6 p.m. to 6 a.m. Urine specimens were taken before getting up, at 6, 7, and 8 a.m. The normal specific gravity for this test was taken as 1,026 or more.

The phenol red test was carried out in 33 patients by the Möller method (Möller and Rex, 1952). The excretion rate was determined after 15 minutes. Normal values: 36% or more.

Urea clearance was determined in 35 patients. Van Slyke's method (McIntosh, Möller, and Van Slyke, 1928) was used. A value of 70% or more was considered normal.

Blood urea was determined on the first, second, or third day of admission by the urease method after King (King, 1953). Normal values: up to 40 mg./100 ml.

The concentration test, phenol red test, and urea clearance test were not undertaken routinely, but were carried out on most patients at various stages of their disease.

Blood pressure was measured on the first day of admission. Normal values: up to 140/90 mm. Hg.

Results

Table 2 gives the types of poisoning, and the laboratory and toxicological findings pointing to the diagnosis of lead poisoning.

The results of the renal function tests and blood pressure findings are summarized in Table 3. The results of repeated analyses are not indicated in this Table.

In Table 4 particulars are given of 27 patients in whom at least one of the tests indicated some pathological changes. The results of repeated analyses are not indicated.

Urine analysis.—In 50 patients urine findings remained normal in repeated examinations carried out at intervals of four to five days during the course of treatment.

Case 1 (Table 4) with pathological urine findings suffered from exudative pleurisy also, which persisted when clinical symptoms of lead poisoning had disappeared. Two years later he was readmitted to hospital with manifest lead intoxication; the urine findings were then repeatedly normal.

Case 2 had repeatedly pathological urine findings in the course of treatment.

Case 3 was readmitted to hospital 10 months after his first treatment, owing to clinically manifest lead poisoning. This time, contrary to the first, the urine analysis was abnormal (albuminuria, erythrocytes, leucocytes, and granular and hyaline casts in the sediment), and remained so three years later when he was again admitted to hospital and treated for lead poisoning.

Blood urea.—In five patients with increased blood urea values in the first few days of treatment (Cases 5, 13, 25, 26, and 27) the tests repeated five to six days later gave normal values.

In Case 2 blood urea values were increased throughout the treatment.

In Case 3 the increased blood urea values became normal in seven days; 10 months later, owing to exacerbation of lead poisoning, he was again in hospital for treatment, showing temporary increase of blood urea values. However, during his third stay in hospital three years later the blood urea values were always raised (140–160 mg./100 ml.).

TABLE 2

CASES OF LEAD POISONING: TYPE AND LABORATORY FINDINGS

	Numbers
Type of Poisoning:	
Gastrointestinal	49
Neuromuscular	3
Encephalopathy	—
Mixed	1†
Laboratory Findings:	
Erythrocytes (10^6/mm.^{3})	
< 3	4
3–4	27
> 4	22
Stippled cells per million erythrocytes	
< 999	—
1000– 2999	8
3000– 9999	19
10000–19999	16
> 20000	10
Reticulocytes per 1000 erythrocytes	
< 3	—
4– 9	1
10–19	9
20–49	19
50–99	20
> 100	4
Lead in blood* (μg./100 ml.)	
< 60	2
61– 99	15
100–199	22
> 200	5
Urinary porphyrins* (μg./100 ml.)	
< 20	—
21– 99	8
100–199	5
200–299	15
300–499	12
> 500	4

27 cases had a lead line on the gums.

* 44 cases.

† The mixed case was a combined gastrointestinal and encephalopathy type.

Blood pressure.—In two (Cases 4 and 24) out of four patients with hypertension (Table 4) blood pressure became normal in five and 10 days respectively.

The blood pressure of Case 2 remained high throughout the whole of his stay in hospital.

Case 5 also remained hypertensive throughout his stay in hospital. At subsequent check-up admissions to hospital two and three and a half years later the blood pressure was also raised (195/105 mm. Hg).

The blood pressure of Case 3 was normal during his first admission, but was increased at the time of

TABLE 3

RESULTS OF THE RENAL FUNCTION TESTS AND BLOOD PRESSURE FINDINGS

Urine		Blood Urea		Urea Clearance Test		Urine Concentration Test		Phenol Red Test		Blood Pressure	
N	P	N	P	N	P	N	P	N	P	N	P
51	2*	46	7	16	19	13	8	12	21	49	4

N = normal. P = pathological.

* One case with proteinuria (opalescence), the sediment containing erythrocytes and leucocytes; and one case with proteinuria +, the sediment containing granular casts.

TABLE 4

PARTICULARS OF 27 PATIENTS IN WHOM AT LEAST ONE OF THE TESTS INDICATED SOME PATHOLOGICAL CHANGES

Case	Age (years)	Occupation	Type of Lead Poisoning*	Urine Analysis	Blood Urea (mg./100 ml.)	Urea Clearance (Per cent)	Urine Concentration Test (Specific Gravity)	Phenol Red Test (Per cent)	Blood Pressure (mm. Hg)
1	52	Peasant	NM	Proteinuria (opalescence) Sediment — erythrocytes and leucocytes	40	57	1025	—	105/60
2	52	Mounting accumulators	GI	Proteinuria + sediment — granular casts	68	8·1	1012	—	230/130
3	49	Peasant	Mixed	Normal	52	26	1020	14·3	130/70
4	24	Manufacture of white lead	GI	Normal	38	81	1022	33·9	160/110
5	52	Lead-mill worker	GI	Normal	42	45·5	—	22·0	185/100
6	34	Mounting electric accumulators	NM	Normal	34	42·5	—	26·9	120/80
7	38	Manufacture of white lead	GI	Normal	38	86	1023	27·8	130/75
8	35	Manufacture of white lead	GI	Normal	40	—	—	24·6	140/90
9	34	Repair of accumulators	GI	Normal	32	72	—	13·9	135/90
10	45	Mounting accumulators	GI	Normal	32	47	1026	33·9	120/70
11	27	Charwoman	GI	Normal	30	43	—	34·0	110/80
12	33	Mounting accumulators	GI	Normal	34	60	—	30·0	130/80
13	36	Packing white lead	GI	Normal	46	56	1028	20·8	130/90
14	33	Mounting accumulators	GI	Normal	37	63·5	—	32·0	130/80
15	25	Mounting accumulators	GI	Normal	38	66·5	—	30·0	120/80
16	28	Lead-mill worker	GI	Normal	40	76	1026	25·6	95/65
17	32	Peasant—potter	GI	Normal	30	52	1026	27·0	115/50
18	26	Mounting accumulators	GI	Normal	38	48·5	1024	12·5	105/80
19	48	Peasant	NM	Normal	40	47·5	1029	30·0	110/80
20	34	Housewife	GI	Normal	35	61·5	1026	20·0	115/70
21	26	Mounting accumulators	GI	Normal	40	43	1026	37·6	130/70
22	22	Manufacture of white lead	GI	Normal	36	81	1022	33·0	130/90
23	43	Mounting accumulators	GI	Normal	32	55	1022	36·4	140/90
24	50	Peasant	GI	Normal	34	48·5	1023	35·1	150/100
25	24	Packing white lead	GI	Normal	48	110	1030	36·5	105/60
26	23	Mounting accumulators	GI	Normal	54	68·5	—	35·0	120/80
27	35	Peasant	GI	Normal	50	—	—	—	135/90

*NM = neuromuscular, GI = gastrointestinal.

his second stay in hospital 10 months later, and became permanently raised by the time of his third stay in hospital after a lapse of three years.

Urine concentration test.—This test indicated pathological changes in eight out of 21 cases examined; it was repeated in a few cases only. The test was repeated in Case 1 whose urine concentration capacity during his first treatment increased to 1,025, and who was readmitted to hospital for treatment for lead poisoning two years later. The results of the urine concentration tests carried out during this second admission are shown in Fig. 1.

The urine concentration capacity of Case 3 in the course of his first stay in hospital increased to 1,020. It only went up to 1,012 three years later, when he was in hospital again owing to manifest lead intoxication.

Phenol red test.—The phenol red test gave abnormal findings in 21 out of 33 patients examined, and was repeated in certain cases. Fig. 2 shows the results of the phenol red test in Case 1 during his second treatment for lead poisoning, two years after the first.

Case 3 repeatedly had decreased phenol red test values after 10 months and again after three years. They were 14·3% (Table 4) during his first stay in hospital and 18·3% and 16% respectively on subsequent admissions.

Case 5 again showed decreased phenol red excretion capacity (11·6% and 24·3% respectively) when examined at routine follow-up after two and

three and a half years, although no symptom of lead poisoning was present.

Urea clearance.—Out of 19 patients with abnormal urea clearance tests (35 patients examined in all) only Case 3 had markedly decreased clearance values at subsequent examinations (39·3% after 10 months, 24% after three years). In Case 24 urea clearance was 48·5% (Table 4) on the seventh day of treatment and 74% on the 33rd day. In Case 5, on his first admission for lead intoxication, urea clearance was 45·5% (Table 4) and after three and a half years 70%.

In Table 4, certain results such as those in Case 25 (blood urea 48 mg./100 ml. and urea clearance 110%) and in Case 26 (blood urea 54 mg./100 ml., urea clearance 68·5%), may appear inconsistent at first sight, but they were obtained at different times; thus in Case 25 blood urea was determined on March 15, 1956 and urea clearance 13 days later, when the blood urea had returned to normal (23·4 mg./100 ml.). There were similar differences in the dates of examination of Case 26.

Case 2 died of uraemia two years after leaving hospital. No autopsy was performed.

Case 3 died during his third stay in hospital. The clinical picture suggested uraemia. Histological examination of the kidney revealed severe tubular lesions with degenerative changes particularly pronounced in the proximal parts of the proximal convoluted tubules. The epithelium of the loops of Henle remained almost completely unaffected. The capsules of some glomeruli were slightly thickened, with patchy hyalinization, while the glomeruli in the subcapsular region were completely obliterated and replaced by hyaline tissue. In the larger intrarenal arteries the intima was thickened, the lumen narrowed, and some sclerotic changes were observed. Small arteries, particularly in the subcapsular region, were hyalinized. The concentration of lead in the kidney was 705 μg./100 g. of fresh kidney, in the liver 1,220 μg./100 g., and in the spleen 482 μg./100 g.

Discussion

On the basis of the results obtained, three facts should be pointed out before any detailed comment is made:

(1) There was a very small number of proved cases of organic nephropathy, and the very small number of pathological urinary findings conflicted with the large number of pathological renal function test values.

(2) Only three out of the 53 patients had a persistently raised blood pressure.

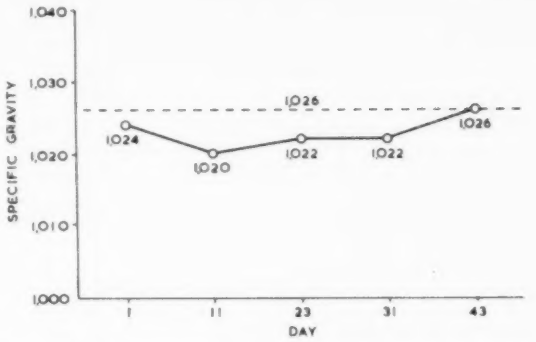

FIG. 1.—Case 1: Urine concentration test results during the second treatment in hospital.

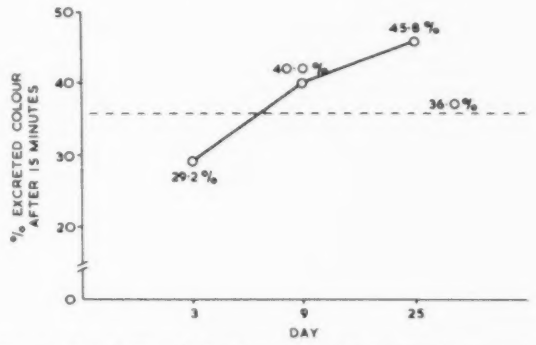

FIG. 2.—Case 1: Phenol red test results during the second treatment.

(3) Some pathological changes returned to normal during the natural course of the disease.

Urine analysis.—Out of three cases with pathological urine findings two were the severest cases in the whole group (Cases 2 and 3), both with well-marked nephropathy; they had had long exposure to lead—22 and 35 years respectively. Case 2 was treated in hospital for lead poisoning on one occasion only, but his history showed that in the course of his exposure to lead he had had several attacks of colic. On his admission to hospital he already had renal insufficiency and, as has been mentioned earlier, died of uraemia two years later. In Case 3 urine abnormalities appeared late, only during his second stay in hospital. Three years later he died of uraemia. It should be pointed out that this patient (a potter !) had been continuously exposed to lead between his admissions to hospital. In the third patient (Case 1) pathological urine findings could not be ascribed to the effect of lead with certainty, as active exudative pleurisy had developed which might have produced albuminuria; in the course of follow-up examinations, when he had recovered from pleurisy, the urine findings

became repeatedly normal, although exposure to lead continued and brought about repeated manifest intoxication. Thus, pathological urine findings, which may have been the result of lead action, were shown to have occurred only in two patients suffering from severe lead nephropathy. These cases confirm the opinion of some writers (Lane, 1949; Vigliani, 1950) quoted above, according to whom severe irreversible nephropathy can be expected to occur in persons with prolonged and intense exposure to lead who have frequently had repeated manifest lead intoxication.

The absence of proteinuria in patients who showed no symptoms of permanent organic kidney lesions is also in accord with the observations of other writers (Radulescu *et al.*, 1957).

In spite of the other pathological findings and a comparatively large number of abnormal renal function test values, the negative urine findings argue decisively against the presence of glomerulitis and other more pronounced lesions of filtration membrane permeability. Thus, on the basis of our observations, the writers (Crepet *et al.*, 1953) who consider glomerulitis and lesions of filtration membrane permeability to indicate the so-called *nephropathia saturnina* cannot be supported.

Blood pressure.—No special comment seems to be necessary about the temporary increase of blood pressure in the phase of manifest lead intoxication. This is a well-known finding believed to be due to the spastic effect of lead on arterial muscle fibres. The fact that only two of our patients showed temporarily raised blood pressure may seem surprising, but this finding would probably have been recorded more frequently if we had had the opportunity to observe the patients from the onset of poisoning.

Of three patients with persistently raised blood pressure, two had severe nephropathy. From the results already given it is seen that in Case 3 the blood pressure rose as the kidney trouble worsened. The third patient with persistently raised blood pressure (Case 5) had no abnormal urine findings. As is seen from Table 4, during his first stay in hospital on account of lead intoxication, the phenol red and urea clearance tests gave diminished values, while the blood pressure was 185/100. During the next two years he was not exposed to lead, but the blood pressure of 180/100 persisted. At this time the urine analysis and the concentration and urea clearance values were normal. Only the result of the phenol red test was worse than that obtained during his first admission. On the third check-up a year and a half later the results were the same. The results of the renal function tests in this patient were identical with those in non-complicated essential hypertension. It cannot be stated that the arterial hypertension and lead exposure in this case (duration of exposure eight years, with repeated attacks of lead colic) were related, but neither can this assumption be rejected on the evidence available.

On the basis of these observations it is suggested that a persistent increase of blood pressure in patients suffering from lead poisoning occurs very seldom. It is usually associated with organic nephropathy which indicates hypertension of a nephrogenic type.

Blood urea.—The permanently increased blood urea levels in two severe cases of nephropathy with renal insufficiency are easy to understand. Mild, temporary azotaemia, observed in some of our patients, has been described in association with lead poisoning (Bell *et al.*, 1925; Radulescu *et al.*, 1957; Hadengue and Collin, 1951). It appears that the earlier the blood urea is measured in a case of lead poisoning, the more likely is azotaemia to be found. In our investigations the blood urea was measured at the beginning of treatment, but had it been examined at the onset of intoxication, *i.e.* had the patients come to hospital earlier, the number of pathological findings would probably have been greater. As lead intoxication advances, the values of N-substances in the blood return to normal in the natural course of events.

Since patients with temporary azotaemia had normal urine findings, there can be no question of any serious glomerular or vascular renal lesions in these cases. As there were no symptoms of the so-called extrarenal azotaemia, some functional intrarenal lesion should be assumed. It may have been a spasm of afferent glomerular arterioles with consequent diminution of the glomerular filtration rate together with temporary azotaemia. The susceptibility of afferent arterioles to lead has been described several times (Crepet *et al.*, 1953; Radulescu *et al.*, 1957; Crepet and Gobbato, 1954).

Urine concentration test.—A diminished urine concentration capacity was frequently found in our patients. It indicates lesions of the distal parts of the tubules, due either to direct toxic effect of lead (Flury, 1934; Beintker, 1929; Radulescu *et al.*, 1957; Clarkson and Kench, 1956; Wilson *et al.*, 1953) or to a reduced intrarenal circulation with consequent hypoxia.

Diminished concentration in the two severe nephropaths seems easy to understand. In other cases there was a mild, probably reversible, diminution of urine concentration (Fig. 1).

Phenol red test.—On the basis of this comparatively inconclusive test no definite conclusion could be drawn concerning selective renal lesions. The results of this test reflect both the excretion capacity of the proximal parts of the tubules and the condition of the intrarenal circulation.

Our investigations have led us to the conclusion that the determination of the amount of phenol red excreted in 15 minutes can reveal more undefined tubular lesions than tests carried out after a longer interval, as in the former case the contact of the dye with the tubular epithelium is shorter.

In Cases 2 and 3 the abnormal phenol red test values can be understood. In Case 5 a reduced phenol red excretion was a permanent functional abnormality. This is assumed to be due to some defect in the intrarenal circulation, as is typical of arterial hypertension.

The repeated phenol red tests in Case 1 (Fig. 2) suggest that in the natural course of the disease abnormal findings may also return to normal (except in cases of irreparable organic renal lesions or persistent arterial hypertension).

The unusual combination of results obtained in the examinations of renal function in Case 24, *i.e.* reduced urine concentration capacity, reduced glomerular filtration rate, but normal phenol red test values can be explained by the fact that the phenol red test was carried out eight days after the two other tests, when renal function may have returned to normal.

These conclusions may seem unjustifiable with regard to the " non-selectivity " of the phenol red test. However, it should be remembered that in addition to a generally recognized diminution of intrarenal circulation rate in lead poisoning (Crepet *et al.*, 1953; Radulescu *et al.*, 1957; Baldi and Sbertoli, 1957), tubular lesions have also been proved to occur (Flury, 1934; Beintker, 1929; Radulescu *et al.*, 1957). These lesions were the dominant feature of the post-mortem findings in Case 4. The selective determination of the excretion capacity of the tubules by para-aminohippuric acid, carried out by various investigators, has also shown that tubular lesions may occur in lead poisoning (Crepet *et al.*, 1953; Rizzo and Sbertoli, 1956).

Urea clearance.—Among patients with abnormal urea clearance values, the lowest were found in two uraemic nephropaths with abnormal urine findings. In other patients the clearance values were slightly or moderately decreased, urine findings persistently normal, while the blood urea values were slightly increased in some patients at the beginning of treatment. We have assumed that the decreased urea clearance values in most patients were due to reduced intrarenal circulation brought about by spasm of afferent arterioles. A decreased filtration rate in lead poisoning has been observed by other writers (Crepet *et al.*, 1953; Rizzo and Sbertoli, 1956). The earlier the examination takes place in the course of intoxication, the more often changes in glomerular filtration rate are observed. Radulescu *et al.* (1957) found diminished glomerular filtration rates in half of 82 patients with lead poisoning. When examinations were undertaken during an attack of colic, no fewer than 70% of the patients showed decreased glomerular filtration rates.

The results of repeated urea clearance tests suggested that the decreased filtration rate was temporary. Persistence or development of glomerular filtration lesions in repeated examinations argue in favour of an organic renal lesion (*e.g.* Case 3).

Most of our patients appear, therefore, to have had decreased glomerular filtration values due to temporary functional disturbances in the intrarenal circulation.

The evidence obtained in this study shows that in most cases the effect of lead upon the kidney manifests itself as reversible changes in the intrarenal circulation and as temporary, slight tubular lesions. These lesions sometimes occurred together. However, owing to rapid changes in these lesions a more thorough insight into their nature can be obtained only by repeated functional examinations in the various phases of lead poisoning, taking into account also the time at which the individual analyses are carried out. By neglecting this point the apparently disparate results of certain renal function tests performed at intervals may obscure the whole picture, and this may indeed have been the cause of many contradictory statements recorded so far on this problem.

Conclusion

On the basis of observations on the effect of lead upon the kidney in 53 patients with proved lead poisoning we have come to the conclusion that this condition can cause renal damage. The changes observed varied widely and we could not confirm the existence of any condition that might be called " *nephropathia saturnina* ". The renal lesions due to the toxic effect of lead may be either functional or anatomical.

Permanent organic changes in the form of chronic progressive diffuse nephropathy with transition into nephrosclerosis and uraemia have been observed only in two out of 53 patients. It should be pointed out that these two patients were just those with the longest and most severe exposure to lead; they were

cases of repeated poisoning. In 23 of the remaining 51 patients various functional renal lesions tending to normalize were observed, without abnormal urine findings.

The data presented are compatible with the explanation referred to in the literature that the partial impairment of renal function is brought about by disordered intrarenal circulation resulting from the spastic effect of lead on the intrarenal blood vessels, and direct toxic or indirect hypoxic effect of lead on the tubules. The occurrence of transitory azotaemia accompanied by normal urine findings may also be explained in this way.

Apart from two cases of temporarily increased blood pressure in the phase of acute poisoning, permanent hypertension appeared to correspond with the development of organic nephropathy. In one case of persistently raised blood pressure of the type of essential hypertension a causal link between the effect of lead and the hypertension could not be proved.

In assessing the results of renal function tests attention should be paid to *when* the tests are made, both as regards the phase of the disease and the intervals between the tests themselves. This is important as the pathological changes do not occur simultaneously nor at the same rate of progress, so that at one moment a certain functional lesion may be present, while at the next moment a different lesion may dominate the picture.

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TRITHION POISONING

BY

C. E. D. HEARN

From Caroni Limited, Trinidad

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Nineteen cases of poisoning by the organophosphorus insecticide trithion (O,O-diethyl *S-p*-chlorophenylthiomethyl phosphorodithioate) are reported among workers on a sugar cane estate in Trinidad. There were no fatalities. Whole blood cholinesterase values determined by a portable field kit are given for some cases. The insecticide was applied as a dust under conditions in which absorption through the skin and inhalation were probably unusually severe.

Cases of parathion poisoning have frequently been described in the literature but there has been no report of poisoning by trithion.

Trithion (O,O-diethyl *S-p*-chlorophenylthiomethyl phosphorodithioate)

is used against the major pest of sugar cane in Trinidad—the froghopper (*Aeneolamia varia saccharina*). Trithion is diluted with limestone dust in mixing sheds to a final concentration of 2% and this dust is applied to the roots of the sugar cane. The published acute toxicity data on trithion show some variability, especially on dermal toxicity. Gaines (1960) quotes an oral LD_{50} of 30 mg./kg. for male rats, and 10 mg./kg. for females; dermal LD_{50} were 54 mg./kg. for male rats and 27 mg./kg. for females. Kerr and Brogdan (1959) quote a dermal LD_{50} of 1270 mg./kg. for the rabbit, and Edson (1960) quotes a dermal LD_{50} of 800 mg/kg. for the rat. The signs of poisoning are those produced by an inhibition of cholinesterase.

Materials

There were 535 workers at risk, of whom 60 were employed in the mixing shed; the rest applied the 2% dust (from "Rotver" dusting guns) to the soil at the base of the sugar cane stool. The men were predominantly East Indian with about 5% negroes.

The whole blood cholinesterase was determined by the rapid field method (Edson and Fenwick, 1955) using a Lovibond Comparator and portable field kit.* All blood samples were taken and estimated in the clinic and not in the field in order to avoid contamination of reagents by drifting clouds of insecticide dust.

Findings

In 1959, 10 cases of trithion poisoning were observed of which seven were mild and two moderately severe and required treatment with atropine. All the cases involved field workers except the one serious case, the circumstances of which are described in detail as it illustrates one of the difficulties involved in supervision.

Case Report.—An East Indian, aged 25 years, was employed in the mixing-shed. This patient was attacked by some of his colleagues during the lunch break, and held down while trithion dust was forced into his mouth. He presented the following morning with nausea, vomiting, severe abdominal colic, excessive sweating, salivation, pallor, apprehension, restlessness, headache, and ataxia. There were fine fibrillary tremors of the lids and face. He required repeated doses of atropine over 30 hours but made a complete recovery.

No cholinesterase estimations were carried out in that year.

In 1960 the working population was surveyed before exposure. The range of normal cholinesterase (ChE) values of 100 men was from 75-87% on the comparator scale (using a European blood as a control 100%). During this season nine cases of poisoning were observed and the clinical features

*Obtainable from Messrs. Tintometer Ltd.

TABLE
NINE CASES OF TRITHION POISONING SEEN IN 1960

Case No.	Date	Race	Age (years)	Clinical Picture	ChE per cent.
1	15.7.60	Negro	18	Anorexia, nausea, vomiting, abdominal colic, pallor, apprehension, restlessness, and headache	37·5
2	15.7.60	East Indian	28	Anorexia, nausea, vomiting, abdominal colic, excessive sweating, salivation, apprehension, and restlessness	37·5
3	20.7.60	East Indian	19	Anorexia, nausea, vomiting, abdominal colic, salivation, and restlessness	25
4	19.7.60	East Indian	22	Anorexia, nausea, vomiting, abdominal colic, excessive sweating, salivation, and bronchoconstriction	37·5
5	22.7.60	East Indian	38	Anorexia, nausea, vomiting, and headache	50
6	22.7.60	East Indian	20	Nausea, slight vomiting	50
7	18.8.60	East Indian	18	Nausea, vomiting, abdominal colic, excessive sweating, salivation, apprehension, restlessness, and headache	25
8	23.8.60	East Indian	21	Nausea, vomiting, bronchoconstriction, and headache	62·5
9	23.8.60	East Indian	28	Nausea, slight vomiting	37·5

together with the ChE levels at the time they were first seen are collected in the Table. Cases 1, 2, 3, 4, and 7 were moderately severe and required treatment. They were kept under observation and given atropine, initially 2 mg. (1/30 g.) intramuscularly, then 1 mg. (1/50 g.) subcutaneously and repeatedly according to their clinical condition (Edson, 1955). All cases made a satisfactory response to treatment and there were no fatalities.

Pyridine-2-aldoxime methiodide (P.A.M.) or the methane sulphonate (P_2S) was not given as these drugs were not available in Trinidad at the time.

Discussion

The hazards involved in the use of some of the organophosphorus insecticides are well known. In Japan it is estimated that parathion leads to about 1,000 cases of poisoning every year (Hiraki and Namba, 1957). Trithion is not so extensively used as parathion and as far as can be ascertained no cases of poisoning by trithion have been previously described. The cases reported here occurred over a three-month period of root dusting during which some 33,695 cwt. of 2% trithion in limestone dust were applied on an estate of 26,000 acres. The application was carried out during the wet season (July-September) when the temperature averaged 84 F. with a relative humidity of 80-90%.

Although those working in the mixing-shed are exposed to a greater concentration of dust, all the cases of trithion poisoning were among the field workers with the exception of the man involved in a fight. The mixing-shed employee works in well ventilated and relatively cool surroundings and is not undergoing arduous physical exertion. Furthermore, the employment of safety precautions can be more easily enforced, at least for most of the time.

The field worker, on the other hand, undergoes strenuous physical exertion as he walks up and down the rows of cane, applying the dust from "Rotver" dusting guns to the roots and raising a dust cloud. The cane is 5 to 6 ft. high acting as an effective shield against any breeze, while strong convection currents tend to maintain the dust cloud. In these intensely hot and humid conditions there is a marked peripheral vasodilation, rapid subcutaneous blood flow, and increased respiratory exchange. These factors facilitate absorption of the insecticide adhering to the skin while the quantity inhaled may be considerable.

The employment of adequate safety precautions in the tropics is a matter of extreme difficulty. Protective equipment is not readily tolerated, and the climatic conditions show their effect also on skin physiology and fatigue. The dusting operations are often of great urgency. The vast majority of the labour force is illiterate and tends either to observe safety instructions with a certain nonchalance, or else to be so impressed by the possible dangers of the work that they will not undertake it at all. The difficulty of ensuring that protective measures are being taken when some of the workers are, of necessity, out of sight of the supervisor as they walk through the cane field during a dusting operation can be appreciated.

Under these conditions an insecticide which can be used safely in temperate climates may carry a hazard in the tropics. The problem is that of supervising all the workers all the time.

Constant vigilance together with the use of the portable field kit makes it possible to detect exposure before serious poisoning develops. It has been found that trithion given in large doses (1-2 g./kg.) will produce ataxia in hens (E. F. Edson, personal

communication). It is satisfactory to note that none of these men developed any delayed neurological lesions.

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A STUDY OF 104 CASES OF MIGRAINE

BY

A. J. CHILDS and M. T. SWEETNAM*

From the Michelin Tyre Company, Ltd., Stoke on Trent

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An inquiry about present or past history of migraine has been made in a factory of 4,700 employees.

The incidence of migraine among the 1,607 who replied to a questionnaire was found to be 4·9% for men and 13·2% for women.

A family history of migraine was found in migraine sufferers six times more frequently than in non-sufferers, and a personal history of travel sickness was found three times more often in sufferers than non-sufferers. It is believed that an association between migraine and travel sickness has not been reported previously.

Migraine was shown to be by no means exclusive to the "intellectual" or "brain-worker" but such people were more often compelled to stop work during an attack than were manual workers.

Treatment taken consisted of analgesics of the aspirin type in 60% of cases and of ergot preparations in 12·5%. The remainder were content to allow the passage of time to terminate their attacks.

Modern treatment, backed by research, has so reduced the severity of many diseases and the time spent in treating them, that other conditions which had previously been regarded as relatively trivial have now assumed greater prominence. Such a condition is migraine, about which much has been written, but nothing of importance has been brought to light since the use of ergotamine tartrate began in the 1920's.

Migraine sufferers, taken to include all those who experience or have in the past experienced attacks of migraine, are stated to comprise 10% of the population; females are affected about twice as frequently as males (Peters, 1953).

The objects of the present investigation were to see whether Peters' figures could be confirmed, to investigate the influence of heredity, and to inquire into other factors, such as the relation of travel sickness to migraine.

Investigation

The present survey was carried out in 1959–1960, at a tyre factory employing nearly 5,000 persons, predominantly male (male to female ratio 4 : 1); 4,700 employees were invited to complete a simple questionnaire and replies were received from 1,607 (34%). The question posed was "Do you now suffer, or have you in the past suffered from migraine?"; migraine was described as "a severe type of headache, usually one-sided, preceded by eye symptoms, usually zigzag lights, and followed by nausea or vomiting".

All those who replied in the affirmative were interviewed and any doubtful cases or definitely negative cases among these were excluded. The criteria used to establish the diagnosis of migraine were the presence of prodromal symptoms, including fortification spectra, scotomata, photophobia, diplopia, and paraesthesiae; headache, nausea, and/or vomiting.

Some otherwise typical cases occurred in which prodromal symptoms were absent and not in all cases did nausea and vomiting occur; these cases were included in the series. All of the employees thus found to have migraine, now or in the past, were then questioned regarding heredity, allergy, travel sickness, treatment, and time lost from work.

Results

Of the 1,607 people who replied to the questionnaire, 1,297 were men and 310 women. After eliminating from the "affirmatives" those who had been mistaken in calling their headaches migraine, it was found that of those who replied to the

* In receipt of the Upjohn Travelling Fellowship of the College of General Practitioners.

questionnaire, 63 of the men suffered or had suffered from migraine, as did 41 of the women. The total number of migraine sufferers was 104 and in both sexes the average age of onset was 23 years, a figure similar to that found by Selby and Lance (1960).

The average duration of attacks in both sexes was one day, and their frequency varied from 30 attacks per year to one attack in three years, with an average frequency in both sexes of eight attacks per year.

Anxiety, fatigue, and excitement were noted as probable predisposing factors to attacks in 39 of the cases. Table 1 shows details of these.

TABLE 1

FACTORS PROBABLY PREDISPOSING TO MIGRAINE ATTACKS

Factors	Men (% of all Affected Men)	Women (% of all Affected Women)
Anxiety (23), fatigue (12), excitement (4)	19 (30·1%)	20 (48·8%)
At week-ends (14) or when relaxed (1)	9 (14·3%)	6 (14·6%)
Bright light (5), noise (4)	3 (4·8%)	6 (14·6%)
Dyspepsia (1), food allergy (4), constipation (1)	3 (4·8%)	3 (7·3%)
Refractive errors (3), bending (1), exposure to cold (1)	4 (6%)	1 (2·4%)

Prodromal symptoms occurred in nine out of 10 of all cases, every female case without exception having prodromal symptoms. Only 12 of the 63 men affected did not experience any such symptoms. Details are shown in Table 2.

TABLE 2

PREVALENCE OF PRODROMAL SYMPTOMS

Symptom	Men (63)	Women (41)	Total (104)
Ocular	48 (76·2%)	39 (95·1%)	87 (83·7%)
Paraesthesiae	3 (4·8%)	2 (4·9%)	5 (4·8%)

Nausea or vomiting or both occurred in 74% of all cases, men 63·5%, women 90·2%.

The influence of heredity was considered and to enable comparisons to be made a random sample was taken from the " negatives ". This was done by taking every 18th name from the alphabetically arranged " negative " replies from men, and by taking every sixth name from the alphabetically arranged negative replies received from women. This method produced a group consisting of 67 men and 44 women, but four men and four women from these lists could not be contacted, leaving the control group to consist of 63 men and 40 women.

The age distribution of this sample compared closely with that of the cases, the average age of the men being 43 years for cases and 45 years in the control group, and of the women 33 years in the cases and 37 years in the controls.

TABLE 3

FAMILY HISTORY OF MIGRAINE AND PERSONAL HISTORY OF TRAVEL SICKNESS IN MIGRAINE SUFFERERS AND A CONTROL GROUP OF NON-SUFFERERS

		Men	Women	Total
Family History of Migraine	Migraine cases	18 (28·6%)	20 (48·8%)	38 (36·5%)
	Control group	3 (4·8%)	3 (7·5%)	6 (5·8%)
Personal History of Travel Sickness	Migraine cases	15 (23·8%)	12 (29·3%)	27 (26%)
	Control group	5 (7·9%)	3 (7·5%)	8 (7·8%)

For the purpose of this investigation a family history of migraine was considered to exist if either parent or any of the sibs of the patient suffered from migraine.

A family history of the condition was found in 36·5% of the migraine sufferers and 5·8% of the controls.

Inquiry was made about travel sickness, in both case and control groups. A personal history of travel sickness was present in 26% of the cases, but only 7·8% of the controls. Details are given in Table 3.

With regard to loss of time from work, it was found that about one-third had to stop work during the attack (men 31·7% and women 36·6%) and that the remainder were able to carry on working, although often at reduced pace or efficiency. All those who stopped work said that they would much sooner carry on if they could, but that the nature of their work, *e.g.* adding figures or typing, made it impossible to do so. Those who continued at work said that it was always much better for them to do so rather than stop. Those in positions of responsibility and in executive grades (classed as managerial, technical, and foremen) were less able to carry on working than those in the less responsible jobs (clerical and light and heavy manual work).

Of the 29 men sufferers who did light manual work, only six (20·7%) had to stop work during an attack of migraine. The figures are shown in Table 4.

TABLE 4

104 CASES OF MIGRAINE: NUMBER WHO STOP WORK OR CONTINUE DURING ATTACKS

Employment Group	Work	Men	Women	Total
Managerial, technical, and foremen	Stop	10	1	11
	Continue	13	1	14
Clerical, heavy, and light manual work	Stop	10	14	24
	Continue	30	25	55

Inquiry was made about loss of time from work due to migraine during the past five years. From the patients' statements it was calculated that the average total of lost time amounted to 141 days per annum. The 63 men had lost an average of 44 days per annum (equivalent to 0·7 day per man per annum) and the 41 women had lost 97 days per annum (equivalent to 2·4 days per woman per annum).

The treatment taken by the patients consisted of some form of analgesic of the aspirin group of tablets in 29 of the women and 34 of the men. Ergot preparations were taken by five women and eight men. The remaining seven women and 21 men took no drugs.

The possible association of the higher incidence of migraine in women with menstruation was considered. Inquiry showed that in 11 women the attacks tended to occur at the time of menstruation. Two women had begun to have migraine with the onset of the menopause.

Conclusions

The overall incidence of migraine found during this investigation in a population with a male to female ratio of 4 : 1 was 6·5% of those who replied to a questionnaire, but this figure should be accepted with reservation as only 34% of the total factory population replied to the questionnaire.

The overall incidence, and the ratio of occurrence in men compared with women is in general agreement with Peters' figures (Peters, 1953). The incidence of menstrual migraine was not sufficient to account for the more frequent occurrence of migraine in women (2·7 : 1) since only in a quarter of the female cases did the attacks occur in association with menstruation.

The figures relating to migraine and heredity showed that in migraine sufferers a family history of the condition occurred over six times more frequently than in the control group who did not suffer from migraine.

Travel sickness also occurred more frequently (3·3 : 1) in migraine sufferers than in non-sufferers. Apparently this association of travel sickness with migraine has not previously been reported.

It is generally believed that migraine affects mainly the intellectual and executive members of the community. This survey showed that the incidence was perhaps higher in this group than in manual workers, as 25% of the cases (Table 4) occurred in the " managerial, technical, and foreman " group, although the total number in this group was a smaller fraction of the whole. Migraine was by no means restricted to this managerial group. What was apparent was that those who had to think and make decisions as part of their work were more often unable to continue than were those doing manual work.

The amount of time lost from work was not found to be great compared with that due to many other major causes of absence. But time lost might be reduced quite considerably from the figure of 141 days per annum if treatment were more specific and prompt.

Few sufferers had found any treatment regularly effective. More than half relied on aspirin-type analgesics, which generally gave only partial relief. The 12·5% who had taken ergot preparations at one time or another did not always have such tablets available for immediate use. Both suffering and time lost could perhaps be reduced by the wider and more ready use of suitable ergot preparations.

The help of Dr. K. Cross of the Dept. of Medical Statistics, University of Birmingham, and of Dr. E. C. Hutchinson, Consultant Neurologist, North Staffs. Royal Infirmary, is gratefully acknowledged.

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BOOK REVIEWS

Occupational Diseases and Industrial Medicine. By Rutherford T. Johnstone and Seward E. Miller. (Pp. vii + 482; illustrated. 84*s*.) Philadelphia and London: Saunders. 1960.

The authors, both physicians and university professors with considerable experience of industrial medicine, have written this book in response to a demand in the U.S.A. It has two parts; the first deals with the practice of industrial medicine and the second with occupational diseases.

Part I includes chapters on the duties of the industrial physician, hygienist, and nurse, various insurance programmes, man's reaction to stress, the rehabilitation of the injured, job placement of the handicapped and the diagnosis of occupational diseases, including the use of the clinical and toxicological laboratory. It will be of interest to the non-American reader because it gives a well written and vigorous account in just over 100 pages of American methods of practice and their development.

The accident and safety phase had its origin in the early years of this century with the passage of workmen's compensation laws. Industrial medicine was concerned almost wholly with traumatic surgery and compensation medicine. With the broadening of compensation laws there came the occcupational disease and industrial hygiene phase which was well organized by about 1930. The present phase of health conservation started at the beginning of World War II, when the U.S.A. suffered a serious shortage of manpower. It became necessary to protect the health of the worker and employ the handicapped and older workers to the best of their capabilities. This is broadly the pattern of development in Great Britain, but in this country there has not been the same stimulus to prevention provided by compensation laws and insurance schemes, and so far there has been less emphasis on industrial hygiene which embodies the measurement of health hazards.

It is quite clear that the good American plant physician has fundamentally the same aims as the good British industrial medical officer. "First and foremost he must be a good clinician and know his plant." "His medical services must have the full backing of management and labour" and so on.

It seems likely, however, that more time is spent in small plants by the physician in the U.S.A. than in Great Britain. "On the average, one hour per day, twice a week, is required for a small establishment of 50 to 75 workers. Establishments of 500 to 600 employees require some physician twice daily." The type of medicine practised is summarized as follows:

First, assistance in placing men in jobs commensurate with their physical and mental abilities. Second, prevention of disease caused by the working environment. Third, practising constructive medicine aimed at improving and maintaining health, particularly keeping emotional tensions from undermining health. And finally, medical treatment for occupational disabilities and treatment of non-occupational emergencies.

Part II, with some 350 pages, contains 13 chapters on occupational diseases and an appendix with the threshold limit values adopted by the American Conference of Governmental Industrial Hygienists. In addition to the classic and common occupational diseases such as the cancers and pneumoconiosis and those caused by noxious gases, hydrocarbons, the metals, ionizing radiations, and physical agents, there are chapters on synthetic resins, plastics, and propellants, and fuels and oxidizers. These are clear and well written chapters and will be valuable to any industrial physician. The sections on treatment are valuable because of the authors' wide experience as clinicians dealing with occupational diseases from discovery to recovery. It is only necessary to disagree with them on points of detail. Most British workers would not agree with the conviction that asbestosis does not predispose to the development of lung cancer or that arsenic is only a suspected cancer producing agent. It is odd, too, to find the statement that the pathology of mill fever is unknown, without reference to the classic studies of Caminita and his colleagues in the U.S.A. Reference might have been made to the International Labour Organization's classification of the Pneumoconioses (*Occup. Safety Hlth* (1959), **9**, 63). This is a good attempt to achieve international uniformity, which, it is stated, has not yet been accomplished.

In the next edition it is hoped that the authors will relate the references at the end of each chapter to the text, and that there will be better pictures of chest radiographs and whole lung sections, even if it makes the book more costly.

This book with its balance of idealism and practicability contains one of the best accounts the reviewer has read of modern methods of industrial medical practice and a good description of the more important and newer occupational diseases. Without any doubt, it will be valuable to industrial physicians both inside and outside the U.S.A.

R. S. F. SCHILLING

Modern Occupational Medicine. 2nd ed. Edited by A. J. Fleming and C. A. D'Alonzo. Associate Editor: J. A. Zapp. (Pp. 587; 66 figures + 1 coloured plate. 90*s*.) London: Henry Kimpton. 1960.

This compact volume on occupational medicine is compiled by 22 authors, all of whom are employed by E.I. du Pont de Nemours and Company of America. It is divided into eight sections, each dealing with different aspects of the industrial physician's work. Lack of space has made it necessary to deal mainly with general

principles, and this somewhat reduces the value of the book to practising industrial medical officers. Brevity is particularly noticeable in the section on toxicology, which is "at best, but a brief outline of some of the more important chemicals encountered in industry". Apart from those subjects normally included in a work on industrial medicine there are a few which are unusual and deserve special mention. A chapter is devoted to the rehabilitation of the alcoholic employee. It does not seem that the condition is as common in England, but the information given will be of great interest to the doctor faced with this problem. The section on psychiatry is stimulating and controversial. The rather stern attitude recommended is a most enlightened and practical approach to the problem of the mentally disturbed patient in industry. The "occupational back" is recognized as a common disability confronting the industrial medical officer, and much useful advice is given in the chapter on this subject.

The book is attractively presented in a style which, although a little unfamiliar to the English reader, is clear and easily understood. The bibliography is extensive but regrettably few references are made to other than American literature. The authors do not set out to make this a work of reference, but it serves as a most useful introduction to modern occupational medicine for both undergraduate and postgraduate students. It can also be recommended to non-medical readers in industry, who wish to know more of the purpose and functions of an industrial medical department.

R. M. OLIVER

Health at Work: A Description of Medical Services in Fourteen British Factories. (Pp. 76.) London: Ministry of Labour and Central Office of Information. 1961.

This book has been prepared by the Ministry of Labour. In a foreword the Minister of Labour emphasizes the importance of promoting health at work from the national standpoint, and hopes that appropriate medical services will be more widely introduced into industrial undertakings.

By describing the health care in 14 different factories, varying much in size and in occupational hazards, this account should encourage employers to assess their own needs and opportunities. Some may need little more than good first aid arrangements and proper attention to plant and office hygiene, with part-time supervision by an experienced doctor or nurse; in others full-time staff with ancillary services such as dentistry, physiotherapy, and chiropody would be more suitable. Even the simplest forms of health care, if provided intelligently with the necessary attention to detail, would be a major advance in much of industry today. The descriptions given here include the views expressed on the services available by the workpeople, managers, doctors, and nurses; these are almost always favourable but perhaps insufficiently critical.

It is notoriously difficult to assess the cost of medical care in a factory and to compare one with another: nevertheless an attempt is made to do so here, the cost varying from 26*s*. to 100*s*. for each employee annually.

This little book, if read by employers considering for the first time the introduction of medical services to their factories, should be helpful, and is therefore to be welcomed.

T. G. FAULKNER HUDSON

Medical Forms and Procedures for Industry. By Herbert L. Herschensohn. (Pp. xv + 192. 68*s*.) Springfield, Illinois: Charles C. Thomas; Oxford: Blackwell Scientific Publications. 1961.

The author has practised industrial medicine in the United States for over 30 years and his book is based on this experience and on the exchange of ideas with others in the same field. It is not his intention to deal with medical treatment but rather with medical policy in (American) industry and the forms most suitable for implementing this policy. Nevertheless, his book will be of more value to the American industrial physician who, unlike his British counterpart, undertakes a considerable amount of treatment. He copes with his task alphabetically and almost literally from A to Z.

The physician who dislikes forms and paper work in general might regard this book as an arsenal of bureaucracy, but there will be few with any experience in industrial health work who will deny the value of a reasoned procedure simplified by the use of ancillary forms.

For the physician about to organize a new industrial health department, or to reorganize one in existence, this book will provide ideas and will help avoid pitfalls, although considerable adaptation will be necessary to suit local circumstances.

It is unfortunate that, in reproducing forms in this book, some print has been reduced to almost microscopic size.

J. L. FYFE

L'Automation: Aspects Psychologiques et Sociaux. By A. Chapanis, A. Lucas, E. H. Jacobson, N. H. Mackworth, L. Ancona, and G. Iacono. (Pp. 119; 10 figures. 110 PB.) Louvain: Publications Universitaires. 1960.

At the 15th International Congress of Psychology a symposium was organized under the direction of Dr. N. H. Mackworth with Professor Drever of the University of Edinburgh as President to discuss some psychological and sociological aspects of automation. This small book includes five papers (four in French and one in English) presented to the Symposium on that occasion. Professor Chapanis of the chair of psychology and industrial engineering at Johns Hopkins, Baltimore in a paper on the human factor in the construction of automation systems shows that automation is very far from being able to dispose of human co-operation and a major problem must be the manner in which the operator is incorporated in such systems. M. Lucas of the Renault Company at Billancourt deals with a concrete situation and suggests that the advent of automation has brought about new tasks which, however, do not differ radically from those which preceded them so that the transformation of the job has been progressive rather than revolutionary. Dr. Mackworth, formerly of the Medical

Research Council Applied Psychology Research Unit, contributes a paper on "Automation and Methods of Research" dealing with laboratory research into the problem and Dr. Jacobson of U.N.E.S.C.O. and Michigan State University one on "Some Socio-psychological Aspects of the Employee's Reactions to Technical Evolution". The concluding paper in English on the same subject by Ancona and Iacono of the Catholic University of Milan deals with observations made in a steel works immediately after automation had been introduced and attempts to assess its effects on the workers. Since there had been a considerable reduction of personnel and at the time of the project the wages of the remaining workers had been reduced one may doubt the value of even the remarkably platitudinous conclusions arrived at which include the remark that "it has not led to a rise in the standard of living or wider economic possibilities".

J. A. C. BROWN

First Aid in the Factory. By Lord Taylor. (Pp. x + 140; 72 figures. 9*s*. 6*d*.) London: Longmans, Green. 1960.

Lord Taylor and his colleagues of the Harlow Industrial Health Service have produced an excellent first aid book, primarily written for the first aider in the factory.

In industry in general and small factories in particular many millions of minor injuries occur every year which are never seen by doctor or nurse, but are treated by first aiders at a first aid box in the workroom, or in a special room set aside for the purpose. Were all these cases to be referred to casualty rooms of hospitals both industry and the National Health Service would grind to a halt. For this reason it is important that first aiders should be trained to recognize minor from major injuries, and be able to treat the minor cases expertly.

After an introduction, the succeeding chapters of this book deal with wounds, haemorrhage, shock, fractures, unconsciousness, etc. in a simple and straightforward manner which can be understood by first aiders. All unnecessary and confusing information has been eliminated, and the 135 pages are liberally illustrated with line drawings which are clear and to the point.

As an example, the mythology of primary, secondary, and other varieties of shock are rapidly disposed of, and the whole subject is dealt with on the basis of fluid loss from the circulation, calling for replacement of fluids by transfusion in a hospital. Hot water bottles are confined to the jumble sale, and drinks are condemned on the grounds that the patient may have to have an anaesthetic.

In the chapter on artificial respiration detailed description is confined to the Holger Neilsen and Eve Rocking Stretcher methods. The mouth-to-mouth method is mentioned briefly but is sensibly not recommended until the position with regard to training is more clearly established.

From these examples it can be seen that here is a practical textbook on first aid, primarily aimed at the vast numbers of men and women who do this work either full or part time in industry, on the farm or on a building site.

It is a book which can be read equally well by doctors who engage in the teaching of first aid, either in or out of industry, more particularly still by those who judge first aid competitions, using out-of-date principles and a plethora of detail which is not only unpractical, but often downright dangerous.

R. A. TREVETHICK

Emergency War Surgery: N.A.T.O. Handbook. (Pp. 411.) Washington: U.S. Department of Defence. 1960.

This excellent little book contains a mass of information concerning the organization and treatment of the war injured. It has been prepared by a committee consisting, among others, of three surgeons, American, British, and French, and represents modern British ideas on war surgery.

Most of the work is based on results from World War II but the war in Korea provided much new data, particularly in regard to vascular surgery. Each chapter contains a reference to the treatment of mass casualties which might result from a future war, when only the less severe injuries could be dealt with.

There are chapters on various types of burns, injuries from cold, and prolonged immersion; other chapters describe chemical and radiation injuries, and new treatment.

Part II is devoted to shock, metabolic disturbances, renal failure, and the treatment of wounds by debridement and antibiotics. Gas gangrene and tetanus are described together with their treatment. It is a little surprising that in the latter no mention is made of continuous anaesthesia.

In Part III wound management is described in detail including sorting and evacuation of the casualties.

Under the section on anaesthesia it is surprising to see spinal anaesthesia mentioned as most surgeons feel this method increases the shock to the injured.

Not many surgeons in this country would agree with the statement "a tourniquet should always be placed about the limb before surgery of the extremity is undertaken".

In Part IV specialized injuries to the head, chest, abdomen, and joints are all well described; all should be moved to special centres as early as possible.

Finally, there is a useful glossary of drugs used in the U.S.A., U.K., and France and a series of conversion tables.

The book is packed with good, sound advice and none of the lessons from the last World War have been forgotten.

G. P. ARDEN

BOOKS RECEIVED

(Review in a later issue is not precluded by notice here of books recently received.)

Toxicology and Biochemistry of Aromatic Hydrocarbons. By Horace W. Gerarde. (Pp. xiii + 329; 89 figures + 63 tables. 30*s*.) Amsterdam: Elsevier. 1961.

Medicina del Lavoro. By Scipione Caccuri. (Pp. xii + 725; 67 figures + 9 tables. L. 9.000.) Napoli: Edizioni Idelson. 1961.

Pneumoconiosis—Modern Trends. Report of meetings held in Birmingham (April 1959) and in Glasgow (January 1960). (Pp. 144; illustrated. 18*s*. 6*d*.) London: The Chest and Heart Association. 1961.

The National Corporation for the Care of Old People. Thirteenth Annual Report for the year ended 30 September, 1960. (Pp. 47.) London. 1961.

Aspects of Public Health Nursing. [Wld Hlth Org. Public Health Papers No. 4.] (Pp. 185. 8*s*. 6*d*.) Geneva: World Health Organization; London: H.M. Stationery Office. 1961.

Expert Committee on Malaria: Eighth Report. [Wld Hlth Org. techn. Rep. Ser. No. 205.] (Pp. 50. 3*s*. 6*d*.) Geneva: World Health Organization; London: H.M. Stationery Office. 1961.

Injuries and Infections of the Hand. By R. H. C. Robins. (Pp. 220; 157 figures. 63*s*.) London: Edward Arnold. 1961.

The Health of Executives, Second Conference. (Pp. 112. 15*s*.) London: The Chest and Heart Association. 1961.

Report on the Health of the Royal Air Force and Women's Services of the Royal Air Force for the Year 1958. (Pp. 52; 21 tables + 10 charts.) London: Air Ministry. 1960.

X-rays—Their Origin, Dosage, and Practical Application, 8th ed. By W. E. Schall. (Pp. xi + 348; 356 figures. 50*s*.) Bristol: John Wright. 1961.

The London Clinic Medical Journal, Vol. 2, No. 1. (Pp. 54; illustrated. 2*s*. 6*d*.) London. 1961.

L'Intoxication Cyanhydrique et son Traitement. By G. Paulet. (Pp. 114; 21 figures + 16 tables. 18 NF.) Paris: Masson et Cie. 1960.

The Practical Management of Head Injuries. By John M. Potter. (Pp. xii + 84. 12*s*.) London: Lloyd-Luke. 1961.

Empire Rheumatism Council. Twenty-fourth Annual Report. 1960. (Pp. 59; 1 figure.) London. 1961.

The Skills of Interviewing. By Elizabeth Sidney and Margaret Brown. (Pp. xii + 396. 35*s*.) London: Tavistock Publications. 1961.

Efficiency and Effort—An Analysis of Industrial Administration. By W. Baldamus. (Pp. viii + 139. 18*s*.) London: Tavistock Publications. 1961.

The Penguin Handbook of First Aid and Home Nursing. By A. C. White Knox and J. E. F. Gueritz. (Pp. 128; illustrated. 3*s*. 6*d*.) Harmondsworth, Middlesex: Penguin Books. 1961.

Tobacco Manufacturers' Standing Committee Research Papers No. 4, Cigarette Smoke Condensate: Preparation and Routine Laboratory Estimation, 2nd ed. By H. R. Bentley and J. G. Burgan. (Pp. 14; 5 figures + 10 tables.) London: Campbell-Johnson. 1961.

Ethiopia in Review, Nos. 12 and 13, Winter and Spring 1961. (Pp. 20; illustrated.) London: Embassy of Ethiopia. 1961.

Central Mining—Rand Mines Group—Health Department. Report for the Year 1960. (Pp. 25; with tables.) Johannesburg: Rand Mines, Ltd. 1961.

The Encyclopedia of Microscopy. Edited by George L. Clark. (Pp. xiii + 693; illustrated. 200*s*.) New York: Reinhold; London: Chapman and Hall. 1961.

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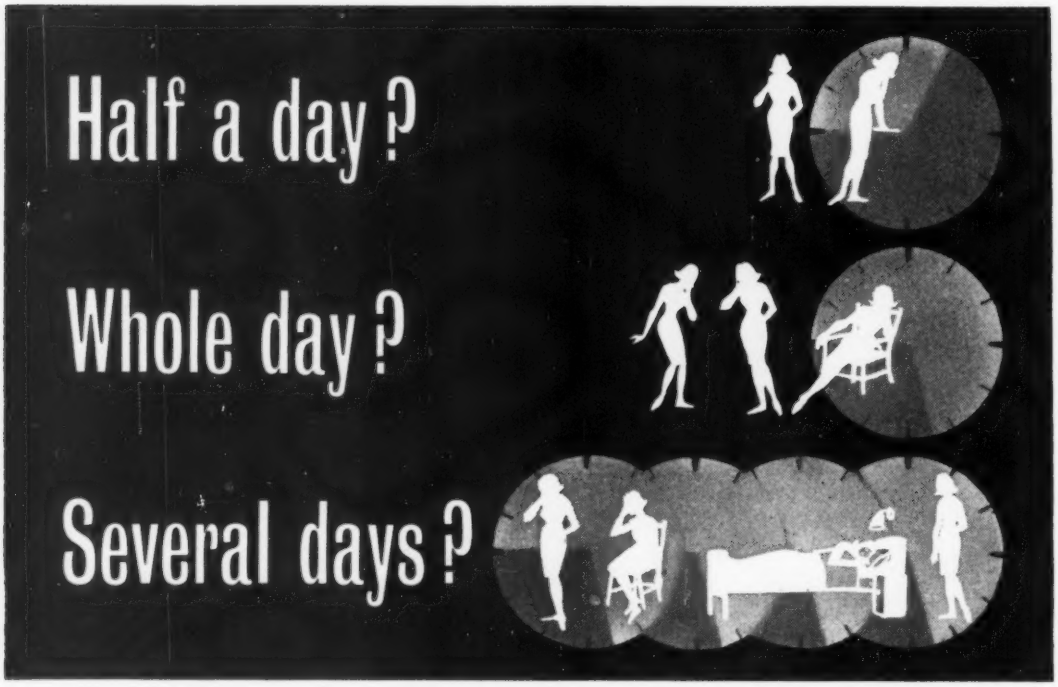

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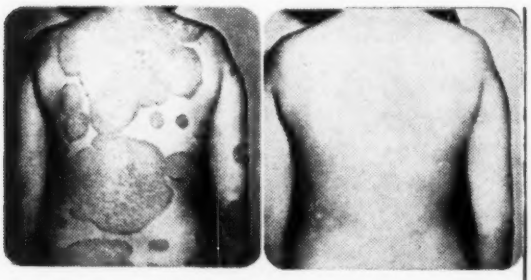

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References: "Clinical Medicine" Vol. 5, No. 4, April 1958: and "Ohio State Medical Journal," Vol. 55, No. 6, June, 1959.

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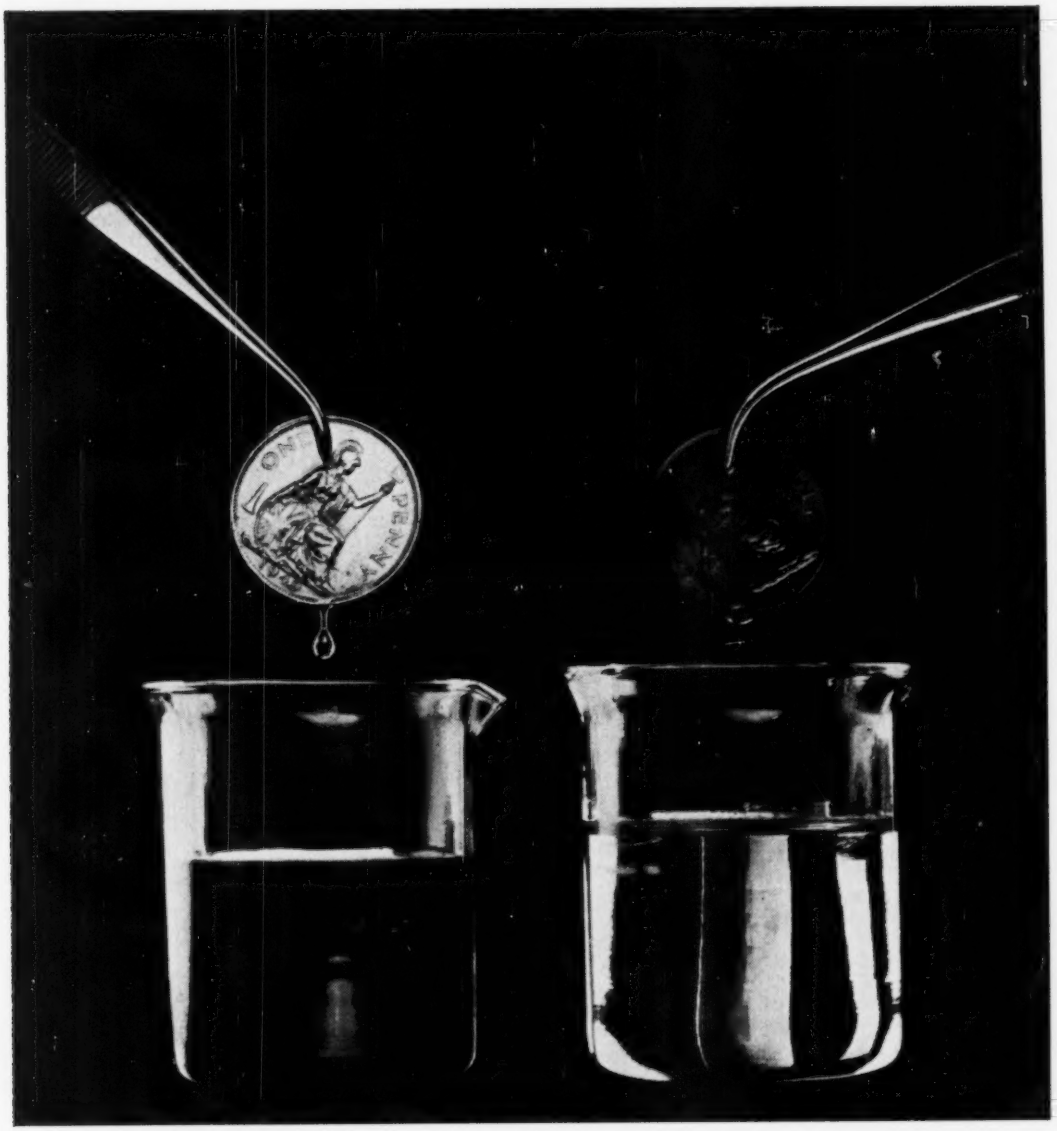

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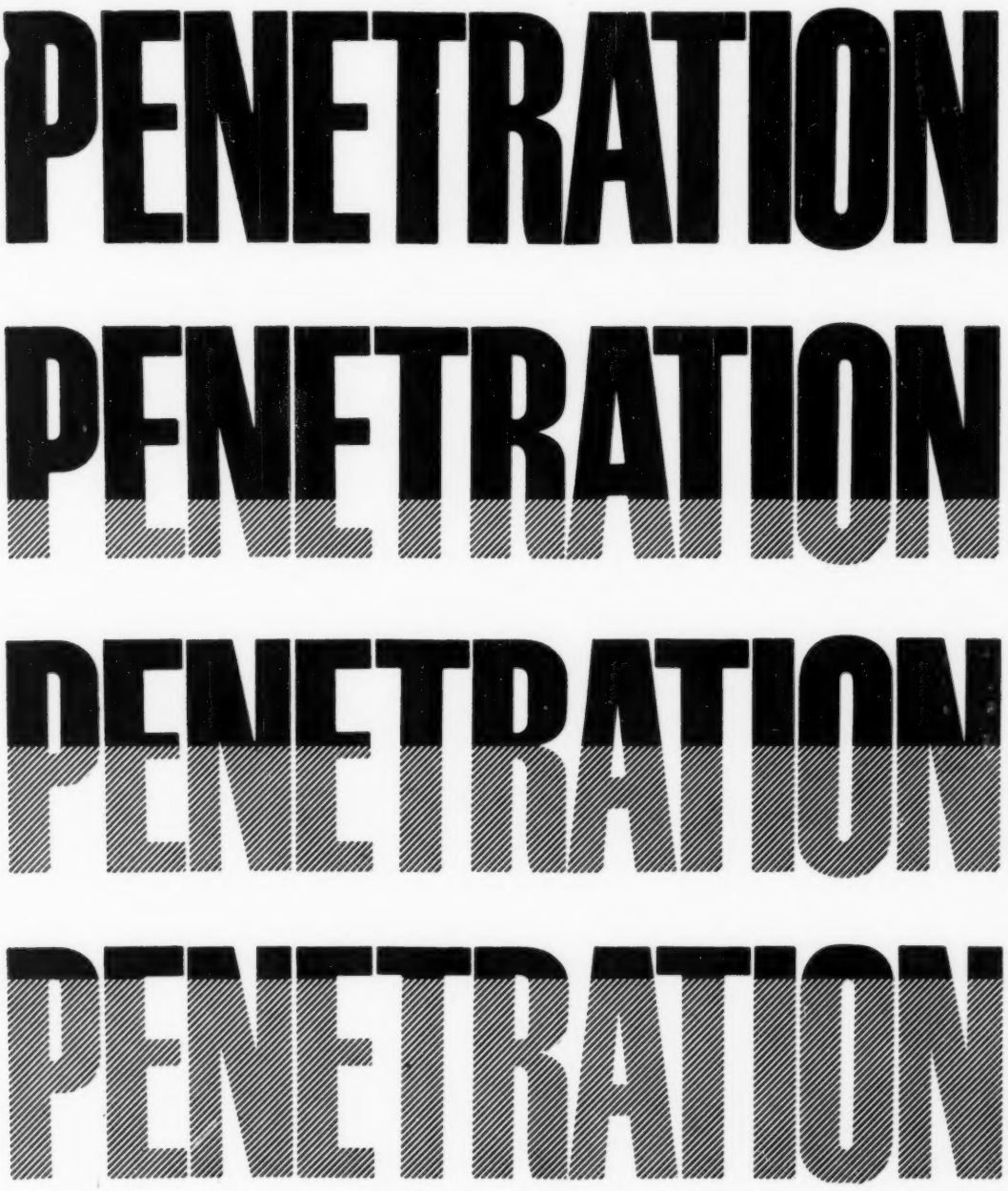

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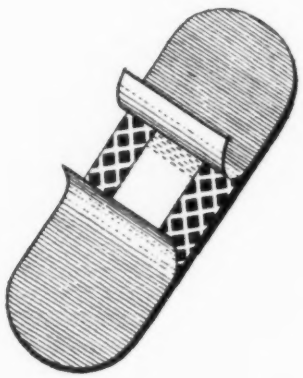

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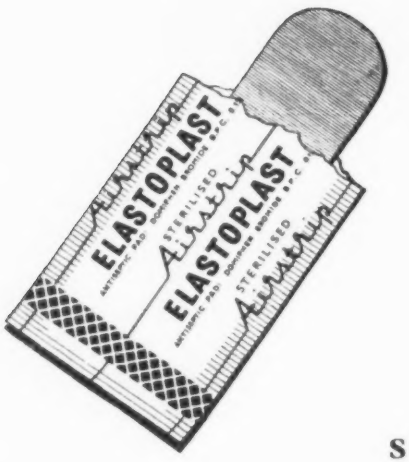

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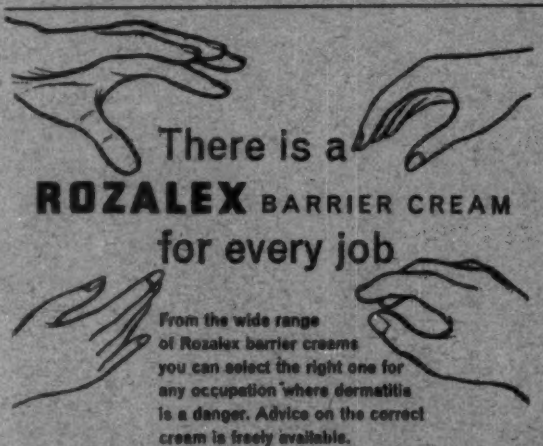
The salary attached to these posts will be £2,000 per annum, in addition to which a rent-free house, motor vehicle and servant will be provided. The applicant will be required to join a provident fund and pension scheme. Thirty days' annual leave is granted and in addition generous long leave accrues at the end of five years' service. Sporting and recreational facilities are provided at all mines. The Company is prepared to assist applicants with passage money to South Africa.

Applications should be addressed in writing with fullest possible details in the first instance to the **Group Personnel Manager, The Cape Asbestos Co. Ltd., 114 & 116 Park Street, London, W.1.**



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